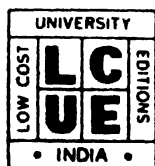
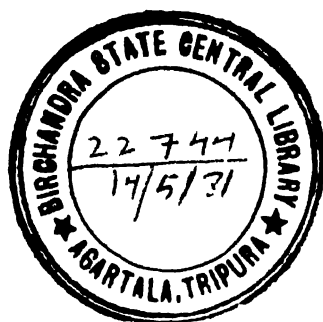


*A Manual of Virus Diseases
of
Tropical Plants*



A Manual of Virus Diseases of Tropical Plants

S P Raychaudhuri



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Foreword

Weather aberrations and disease epidemics constitute two of the most important factors of instability in crop production. Among diseases those caused by viruses are some of the most devastating and difficult ones. Seed-borne viruses and virus diseases of vegetatively propagated plants in particular cause considerable loss as well as complications in quarantine arrangements. Some of the virus diseases like tungro and grassy stunt of rice leaf curl of tomato, tobacco and chilli and yellow mosaic of pulses and vegetables cause varying degrees of damage in different parts of our country. Certain complex diseases like citrus dieback and coconut root will have a virus component in them. More intensive research on all aspects of virus diseases is hence a growing necessity.

Dr. S.P. Raychaudhuri is to be congratulated for undertaking the task of compiling all the available information on important virus diseases with special reference to tropical areas. Over 250 virus diseases are described in this manual along with over 100 black and white as well as coloured illustrations. Viruses (not virus diseases) are referred to using popular English names thereby avoiding the controversial field of plant virus nomenclature. Dr. Raychaudhuri has brought the book up-to-date by having a separate chapter on diseases caused by mycoplasma-like organisms. Thus, this comprehensive manual will be of immense use to all research scientists and students interested in plant virology. This manual is another index of the dedication of Dr. Raychaudhuri to the cause of promoting virus research in our country.

M.S. Swaminathan, F.R.S.
Director-General, ICAR and
Secretary to the Government of
India, Ministry of Agriculture.

NEW DELHI

Preface

More than 30 years ago I got interested in plant virus diseases when I had the opportunity to work under the distinguished leadership of Dr. B.N. Uppal, formerly Plant Pathologist to the Government of Bombay and also Agricultural Commissioner with the Government of India. Later, while working at the Rockefeller Institute (now Rockefeller University) in New York alongwith outstanding and internationally known scientists like the late Dr. L.O. Kunkel, Dr. F.O. Holmes, Dr. Karl Maramorosch and Dr. Russel L. Steere. I got a lot of inspiration and guidance for conducting plant virus research. There used to be a lot of discussions about viruses infecting tropical plants. During several discussions with the late Sir Frederick C. Bawden and Dr. Kenneth M. Smith of the U.K., I was very much influenced by their ideas specially in researches on applied plant virology and the necessity of writing a book on virus disease of tropical plants.

While working at the Indian Agricultural Research Institute I was encouraged by Dr. M.S. Swaminathan, the then Director of the Institute, now Director General of the ICAR, New Delhi who had been kind enough to readily agree to render the necessary financial support for preparing this book. I am grateful to all the persons mentioned above as well as the University Grants Commission for the encouragement received from them. I have separately acknowledged the help I received from various friends and colleagues abroad as well as in India for helping in various ways in completing the book.

It is very gratifying that the National Book Trust of India also kindly agreed to give a subsidy so that the book will be sold at the cheaper rate for the benefit of scientists, teachers and students.

S P Raychaudhuri

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I must make mention of the photographers who helped me in various ways.

I express my gratitude to **Mrs. D. Rajyalakshmi Rao, Miss R. Padma, Sh. S.M. Viswanath, Dr. Atin Ghosh, Miss Mahua Duttagupta, Dr. V.S. Varma and Mrs. Prasannakumari Pillai** for helping in some final corrections.

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SPR

Contents

Foreword	v
Preface	vii
Acknowledgements	ix
List of Plates	xiii
List of Colour Plates	xix
Introduction	1
1. Symptomatology	8
2. Transmission	12
3. Physiology of Infected Plants	25
4. Purification of Plant Viruses	36
5. Serology	43
6. Detection at Seed and Cellular Level	52
7. Tissue Culture	61
8. Virus Inhibitors	69
9. Control	81
 VIRUS DISEASES	
10. Cereals	92
11. Fibre Crops	114
12. Forage Crops	121
13. Forest Trees	124
14. Fruits	126
15. Medicinal Plants	153

16. Millets	157
17. Oil Seeds	162
18. Ornamentals	173
19. Plantation Crops	189
20. Pulses	210
21. Spices	220
22. Vegetables	224
23. Weeds	263
24. Diseases Attributed to Mycoplasma and Rickettsia-like Organisms	268
Index	293

List of Plates

All plates appear between pages 188 and 189

- Plate 2.1 (a) Non-persistent Virus
(b) Persistent Virus
- Plate 2.2 Leafhopper *Cicadulina mbila* vector of bajra streak virus feeding on bajra plant
- Plate 7.1 Apple tumor virus in tissue culture
Right: Callus from tumor tissue
Left : Callus from healthy tissue
- Plate 7.2 Culture of exocortis affected citrus bud on MS medium
- Plate 7.3 Differentiation of shoots in stem callus tissue of *Citrus grandis*
- Plate 7.4 Healthy sugarcane plant raised by meristem culture
- Plate 10.2 Vein enation of maize
- Plate 10.3 Vein enation of maize on wheat
- Plate 10.4 Vein enation of maize on rice
- Plate 10.5 Vein enation of maize on ragi
- Plate 11.1 Yellow mosaic of jute
- Plate 11.2 Sunnhemp mosaic
- Plate 11.3 Electron micrograph of Southern Sunnhemp mosaic virus
- Plate 14.1 Apple mosaic
- Plate 14.2 Apple star crack

- Plate 14.3 Apricot mosaic
- Plate 14.4 Banana Mosaic
- Plate 14.5 Banana bunchy top
- Plate 14.6 Cape gooseberry mosaic due to CMV
- Plate 14.7 Cape gooseberry mosaic due to TMV
- Plate 14.8 Cherry mosaic
- Plate 14.9 Tatter leaf of cherry
- Plate 14.10 Leaves of lisbon lemon seedling showing spots when inoculated with lemon crinkly leaf virus
- Plate 14.11 Leaves of eureka lemon affected with lemon crinkly leaf virus
- Plate 14.12 Cracking of bark and elongated lesions by exocortis on Rangpur lime
- Plate 14.13 Cracking of bark by exocortis on sweet lime
- Plate 14.14 Exocortis affected grape fruit trees
- Plate 14.15 Citrus tristeza (affected sweet orange tree on sour orange rootstock on right)
- Plate 14.16 Flexuous rods from orange tree (*Citrus sinensis*) infected with tristeza virus
- Plate 14.17 Citrus tristeza virus on Kagzi lime (*C. aurantifolia*)
Healthy leaf on right
- Plate 14.18 Stem pitting symptoms of tristeza virus on *C. aurantifolia*
- Plate 14.19 Fig mosaic
- Plate 14.20 Mulberry mosaic
- Plate 14.21 Mulberry yellow net-vein
- Plate 14.22 Papaya leaf curl
- Plate 14.23 Plum line pattern
- Plate 16.1 Ragi mosaic
Left : Healthy leaf
Right : Diseased leaf
- Plate 16.2 Electron micrograph of *Ragi* mosaic virus

- Plate 16.3 *Bajra* mosaic
- Plate 16.4 *Bajra* streak virus on wheat
- Plate 16.5 *Bajra* virus on barley
- Plate 17.1 Groundnut mosaic
- Plate 17.2 Groundnut ring spot
- Plate 17.3 Groundnut bunchy top
- Plate 17.4 Groundnut chlorosis
- Plate 18.1 Canna mottle
- Plate 18.2 Electron micrograph of Canna mottle virus
- Plate 18.3 Dahlia mosaic virus
 Right: Infected dahlia
 Left: Uninoculated control
- Plate 18.4 Dahlia mosaic virus on dahlia showing chlorosis
 along the veins
- Plate 18.5 Section of leaf of *Zinnia* infected with Dahlia mosaic
 virus - 40,000
 V — Virus particles G — Golgi complex
 W — Cell wall
- Plate 18.6 *Hippeastrum* leaves affected with mosaic diseases
 Left: Healthy leaf
 Right: Diseased leaf
- Plate 18.7 Mosaic of *Tropaeolum majus*
- Plate 18.8 Mosaic of *Vinca rosea*
- Plate 18.9 Yellow vein mosaic of *Rosa bourbina*
- Plate 18.10 *Hibiscus rosa-sinensis* leaf curl
- Plate 19.1 Cassava mosaic
- Plate 19.2 Coconut root (wilt)
- Plate 19.3 Electron micrograph of coconut (root) wilt virus
 × 30,000)
- Plate 19.4 Mosaic of sugarcane

- Plate 19.5** Long flexuous rod-shaped particles of sugarcane mosaic virus $\times 60,000$
- Plate 19.6** Tea rose yellow mosaic
- Plate 19.7** Tea phloem necrosis
- Plate 19.8** Tobacco broken ring spot
- Plate 19.9** Tobacco etch virus
- Plate 20.1** Healthy and sterility affected pigeon pea plants
Left: Healthy plant bearing flowers
Right: Diseased
- Plate 20.2** Pigeon pea sterility mosaic
Left: Healthy leaf
Right: Diseased leaf
- Plate 20.3** Yellow mosaic of mung (*Phaseolus aureus*)
- Plate 20.4** Soybean mosaic
- Plate 20.5 A** *Aceria cajani* the eriophyid mite vector of pigeon pea sterility virus
- Plate 21.1** Katte disease of small cardamom
 (a) Healthy leaf (b) and (c) Diseased leaves
- Plate 21.2** Chirke disease of large cardamom
- Plate 21.3** Large cardamom affected by Foorkey virus
 (a) Foorkey infected plant (b) Healthy Plant
- Plate 22.1** Beet root purple top
- Plate 22.2** Mosaic of bean (*Phaseolus vulgaris*)
- Plate 22.3** Yellow vein mosaic of *Bhindi* (Okra)
Left: Healthy leaf
Right: Diseased leaf
- Plate 22.4** Mosaic of chilli
- Plate 22.5** Cucumber mosaic (Cucumis virus 1)
- Plate 22.6** Snakegourd mosaic (Cucumis Virus 1)
- Plate 22.7** Electron micrograph of CMV (Cucumis virus 1)
- Plate 22.8** Bottlegourd mosaic (Cucumis virus 2)

- Plate 22.9 Watermelon mosaic (Cucumis virus 2)
- Plate 22.10 Electron micrograph (Cucumis Virus 2)
- Plate 22.11 Pumpkin mosaic (Cucumis virus 3)
- Plate 22.12 Mosaic of *Cucurbita pepo* (Cucumis virus 3)
- Plate 22.13 Electron micrograph (Cucumis virus 3)
- Plate 22.14 Lettuce mosaic
- Plate 22.15 Mosaic of garlic
- Plate 22.16 Potato mosaic
- Plate 22.17 Leaf roll of potato
- Plate 22.18 Veinal necrosis induced by potato virus Y in potato
- Plate 22.19 Enation leaf curl of tomato
- Plate 24.1 Apple rubbery wood
- Plate 24.2 Greening-affected mandarin trees
- Plate 24.3 Grape fruit affected with greening showing defoliation of leaves
- Plate 24.4 A leaf of greening affected sweet orange showing yellowing of midrib and lateral veins
- Plate 24.5 Greening affected kagzi lime
- Plate 24.6 *Diaphorina citri*, Kuway, the vector of citrus greening
- Plate 24.7 Fried egg-shaped colony of citrus greening mycoplasma on PPLO agar medium
- Plate 24.8 Small-leaf disease of cotton
Left : Healthy
Right : Diseased
- Plate 24.9 Mycoplasma-like bodies in the phloem sieve cells of small-leaf affected cotton leaf
- Plate 24.11 Marginal flavescence of potato
- Plate 24.12 Witches' broom disease of potato
- Plate 24.13 Mycoplasma-like bodies in the potato witches' broom affected cells
- Plate 24.15 A : Spiked sandal tree
B : Healthy sandal tree

C : MLB in plant cells under electron microscope

D : Little-leaf affected brinjal plant

Plate 24.16 **Diseased sandal shoot after treatment.**

Note: appearance of healthy leaves

Plate 24.17 **Sugarcane grassy shoot**

Plate 24.18 **Mycoplasma-like bodies in the sieve tubes of sugarcane affected with grassy shoot**

Plate 24.19 ***Sesamum* affected with phyllody**

Plate 24.20 **Mycoplasma-like bodies (M) in phloem cells of *Sesamum* affected with phyllody**

List of Colour Plates

- 6.1 Detection of citrus greening by fluorescent antibody technique. Stained mycoplasma-like bodies in phloem show apple green fluorescence.
- 6.2 Detection of Cowpea mosaic virus in cowpea leaves by fluorescent microscopy. Epidermal peelings show virus inclusions adjacent to the nucleus.
- 6.3
 - a. Detection of Cowpea mosaic virus in seed by fluorescent microscopy. Cells from diseased seed showing intense red fluorescence.
 - b. Cells from healthy seed
- 6.4 Detection of coconut root (wilt) by aerial infra red photography. Trees in circles show severe disease intensity.
- 10.1 Maize Mosaic.
- 10.6 Bajra streak.
- 10.7 Rice tungro virus.
- 10.8 Mosaic streak of wheat.
- 24.10 Peanut withches' broom.
- 24.14 Rice yellow dwarf.

Introduction

Plant viruses were known long before the discovery of bacteria; 'tulip breaking' being one of the earliest records reported by Charles l'Ecluse of Carolus Clusius in 1576. However, the first scientific proof of the existence of virus disease in plants was provided in 1892 by Iwanowski, a Russian botanist, and was confirmed in 1898 by Beijerinck. Since then the science of plant virology has advanced very rapidly and the last seventy-five years have seen enormous progress of this important branch of plant pathology. During the first quarter of the twentieth century most attention was paid to symptomatology, identification and methods of transmission of plant viruses without knowing the nature of the causal agent as it could not be seen due to limitations of the light microscope. After the first isolation of tobacco mosaic virus in crystalline form by Stanley in 1935, attention was paid to the study of the causal agent, its isolation in purified form, morphology and chemical composition. The invention of the electron microscope made possible to study further the shape and size of virus particles.

In higher plants the shapes of viruses range from spheres to rods or thread-like structures of various sizes. The isometric viruses usually appear spherical in shadowed preparations but many, are polyhedral. The anisometric ones fall into three readily distinguishable categories (1) Bacillus-like or bullet shaped with rounded ends and varying dimensions, e.g., alfalfa mosaic and mushroom viruses, (2) rigid rods such as Tobacco Mosaic Virus (TMV) and tobacco rattle virus, (3) flexible threads, e.g., potato virus Y or citrus tristeza virus.

On the basis of electron-microscopic and X-ray diffraction studies, it has been shown that most of the plant viruses with a few exceptions have their outside surfaces composed of regularly arranged protein units with their nucleic acid carried internally. The type of pattern in which the protein sub-units are arranged

differs with different viruses as also the position and orientation of nucleic acid relative to the protein covering. In rod-shaped viruses like tobacco mosaic virus the protein sub-units are spirally or helically arranged around a hollow core. The spherical or polyhedral viruses are on the other hand shells of protein enclosing nucleic acid. Most of the plant viruses contain ribonucleic acid (RNA). Some plant viruses containing deoxyribonucleic acid (DNA) have been reported in the recent years, such as, cauliflower mosaic, carnation etched ring and dahlia mosaic. Few cases of plant viruses, namely, potato spindle tuber, citrus exocortis and chrysanthemum stunt have been found to exist in the form of naked nucleic acid for which the term 'viroid' has been used. Besides, a common group of diseases known as the 'yellows' type of diseases characterised by general yellowing, stunting, witches' broom-like growth and phyllody have been associated with mycoplasma-like organisms (MLO).

With the progress of research on biophysical and biochemical properties of plant viruses, advances have also been made in other related fields such as modes of transmission, vector relationships, serology and methods of detection involving staining techniques. Potent vectors among nematodes, mites and fungi have been discovered. Techniques like fluorescent antibody and infra-red photography have been developed for early and quick detection of viruses.

The control of plant viruses has received particular attention. Heat and chemotherapy have been used in several cases to inactivate viruses in plants and planting materials. The importance of seed certification has been realised and certification programmes have been recommended for a number of fruits and vegetatively propagated crops like citrus, temperate fruits, potato, etc. Direct methods of control have been used for control of vectors by systemic insecticides and oil sprays to reduce their spread, although this has its own limitations. The best and cheapest method of control of plant virus diseases is by the use of resistant varieties. However, in practice it is not always possible to get genes in resistance variety alongwith other desirable characters. Hence the struggle for finding alternate methods of control will continue so long as the viruses constitute a danger to our cultivated crop plants.

It is not possible to give a detailed account of all the aspects of plant virology in a short account like this which is primarily

designed to give a detailed account of the diseases occurring in tropical region rather than to give details of principles of plant virology. Only the work done on these aspects in tropics has been dealt with. For details on principles of plant virology readers are advised to read standard textbooks.

Symptomatology

More than eighty years have elapsed since Adolf Mayer coined the term, 'mosaic', to describe a transmissible disease of tobacco. Ever since, this term has become very popular and is being widely used when there is an uneven distribution of development of chlorophyll causing light green or yellow areas on leaf lamina. This and many other like manifestations in plants can generally be related to disturbed plant metabolism usually caused by viruses and such other pathogens.

Presence of a virus is generally recognised by its symptoms on the host. At one time, symptoms were the only means of detecting virus diseases in plants. Most viruses are named on the basis of chief symptoms produced on the hosts on which they were first reported, e.g., potato leaf roll virus, tobacco leaf curl virus, etc. Now it is well known that symptoms alone cannot characterise a virus since they vary under different climatic conditions and some symptoms on the same host may be developed by unrelated viruses.

Symptoms of virus diseases may range from no symptoms to different degrees of severity. Most of the viruses are generally systemic in their host plants and they take time in moving to other tissues. Therefore, in large trees or woody plants, symptoms may appear early in some branches than in others. Also, in most cases the mature leaves at the time of initiation of infection rarely show symptoms. The first symptoms are usually seen in young growing leaves at or near the growing point. Sometimes the viruses produce localised reaction called the local lesions. A broad outline of the symptoms which are commonly associated with virus diseases are given below to facilitate the readers to get an idea of such disorders.

The symptoms could be broadly classified into two major groups (1) *External*—which could be seen by naked eye (2) *Internal*—that is cytological and histopathological changes brought by viruses in the tissues of the plant.

EXTERNAL SYMPTOMS

Local

Certain plants manifest a reaction restricted to the locus of infection upon inoculation with a virus. Such local lesions are only important for biological assay. Based on their reaction the lesions may be characterised as under:

Chlorotic lesions: They are formed when the cells in localised areas around the site of infection lose their chlorophyll and other pigments. The lesions may be pale to almost white, e.g., sugarbeet mosaic virus on *Chanopodium amaranticolor*.

Necrotic lesions: The affected leaf area becomes characterised by dead cells, the extent of necrosis varying in size and shape from a small pinhead (as TMV on *Nicotiana glutinosa*) to as large as five mm in diameter (spherical cowpea mosaic on *Chenopodium amaranticolor*). The lesions may appear chlorotic in the beginning but they turn necrotic.

Ring spots: Ring spot type of lesions are formed when concentric group of chlorotic or necrotic and normal green tissues alternate as those formed due to tobacco ring spot virus on *Nicotiana rustica*.

Systemic

Stunting: It may affect all parts of plant, involving a reduction in the size of leaves, flowers and fruits and a shortening of petioles and internodes, e.g., bean yellow mosaic virus on bean. Extent of stunting varies with the developmental stage of the plant when the infection occurs. Sometimes a few parts may be considerably more stunted than others.

Colour changes in the foliage: They may range from faint chlorosis to prominent vein line patterns, as in case of yellow vein mosaic on *bhindi*. The other type which is of common occurrence is the mosaic pattern which is generally irregular in outline and the borders between darker and lighter areas may not be distinct. In some mosaic diseases the dark green areas are associated mainly

with the veins to give a vein banding pattern as in case of cauliflower mosaic virus.

In monocotyledonary plants virus infection usually produces chlorotic stripes or streaks of varying sizes on the leaves. The stripes or streaks run parallel to the length of the leaf as seen in *bajra* streak.

Changes in leaf form: Uneven growth of leaf lamina is often found in mosaic diseases. Leaves become curled (curling), brittle (crinkling) and show prominences and depressions (puckering). Upward and inward rolling of leaf is sometimes very prominent, e.g., potato leaf roll. Frequently the lamina gets extremely reduced giving 'fern leaf' effect (filiform), e.g., TMV or CMV on tomato. In cherry leaves, the lamina develops perforations giving 'tatter leaf' appearance.

Ring spot: The infection in many virus diseases results in a pattern of concentric rings and irregular lines on the leaves and sometimes also on the fruit, e.g., tobacco ring spot. Lines consist of chlorotic tissues or may be due to death of superficial layers of cells.

Necrosis: Infection of leaves may develop scattered flecks or patches of dead tissue. Necrotic pattern may follow the veins as the virus moves into the leaf, e.g., tobacco necrosis.

Wilt: In some cases, the virus infected plants exhibit yellowing and dropping of the outer whorls of leaves and also inner whorls become pale yellow in colour. The leaflets soon turn brown in colour and start drying up from their tips. The leaves start shedding in quick succession after a plant is infected. The leaves become smaller and progressively stunted in size. There is also considerable reduction in root growth, e.g., coconut root wilt disease (Nagaraj and Menon, 1956).

Production of outgrowths: Abnormal growth or malformation in virus diseased plants is common. Enation or foliar outgrowths and vein swelling, etc., are generally formed on underside of the leaves of infected plants, e.g., tobacco leaf curl disease, Fiji disease of sugarcane, etc. The formation of tumors or swelling on stems and roots are also met with as in case of sweet clover infected with wound tumor virus, cocoa infected with swollen shoot virus and woody gall of citrus.

The other symptoms are bark scaling, stem pitting, and bark cracking due to various citrus viruses and rubbery wood due to a virus infection in apple.

Flower breaking: Breaking of flower colour is quite common in virus infections, e.g., tulip breaking. In fact this is the oldest known virus disease symptom and is of commercial value.

Phyllody, sterility and witches' broom: These symptoms are characteristic of yellows type of diseases most of which have recently been associated with mycoplasma like bodies. The most common symptom is the virescence or greening of floral parts and stimulation of axillary buds to give bushy appearance to the plants, e.g., sesamum phyllody.

Fruit abnormalities: Fruits produced on virus infected plants may show a variety of symptoms, e.g., mottling on cucumber and papaya due to mosaic type of viruses and star crack in apples and plum, pox in plums due to plum pox virus. Fruits may become acorn-shaped as in citrus stubborn mycoplasma disease.

DISEASE RECOVERY

It is commonly noticed with certain viruses that plants show disease symptoms for some time and later on new growth symptoms are milder or absent, although virus is still present, e.g., tobacco plants infected with sugarbeet curly top. Potato plants show recovery from mosaic symptoms due to potato virus X in summer months.

INTERNAL SYMPTOMS

Several cytological and histological abnormalities can be observed within the virus affected plants. The histological deviations, although especially seen in tissues, are actually of purely cytological origin.

Influence of viruses on the shape of cells is not well known but the effect of viruses on the size and the number of cells is observed. When there is an abnormal increase in the size of cells, the condition is called 'hypertrophy'. Abnormal increase or decrease in the number of cells is referred to 'hyperplasia' or 'hypoplasia', respectively. Leaves with mosaic symptoms frequently show hypoplasia in the yellow areas. The lamina is thinner than in the dark green areas. The cells of mesophyll are less differentiated with fewer chloroplasts and fewer or no intracellular spaces. The unlimited increase in number of cells is termed as cell proliferation. In swollen shoot disease of cocoa, abnormal

amounts of xylem tissues are produced in shoots but the cells appear structurally normal (Posnette, 1947).

In case of some viruses at least vein clearing symptoms are due to enlargement of cells near the veins (Esau, 1956). Due to the presence of less chlorophyll in the tissues the leaf may become more translucent.

Another term, atrophy, is used for completely arrested development of cells or organs and for a total lack of cell multiplication and cell enlargement.

In potato leaf roll disease, the problem develops normally but is killed due to necrosis. In sugarbeet curly top, there is degeneration of phloem accompanied by supernumerary sieve tubes (Esau, 1956). In other cases necrosis may start in the phloem and spread rapidly to other tissues, e.g., top necrosis of potato. Potato virus Y involves necrosis beginning in the leaves and spreading along the veins, mainly in collenchyma tissue without the vascular elements being affected (Bawden, 1932).

A major cytological effect of virus infection is the development of inclusion bodies visible by light microscopy in infected cells. There are two main types of inclusions, crystalline and amorphous; the latter are also known as X-bodies. Beale (1937) observed that the crystalline inclusions in TMV closely resembled the paracrystals of TMV first isolated by Stanley (1935). These inclusion bodies commonly occur in epidermal cells of leaves and stem, but they also occur in the roots and flowers and in most tissues except in the phloem and sieve elements (Bawden, 1964). Sheffield (1946) showed that the X-bodies caused by tomato aucuba mosaic contained large amounts of virus. She also showed by means of electron microscopy that they contained many virus rods. Matsui and Yamaguchi (1964a) studied X-bodies associated with other viruses. X-bodies contain dense peripheral zone and an internal matrix, e.g., Tobacco etch virus on *Datura stramonium*. Cells of *Pisum sativum* infected with red clover vein mosaic virus show X-bodies which contain coiled filamentous particles (Rubio-Huertos, 1964). X-bodies seen in epidermal stripes of plants infected with broad bean mottle virus and cabbage black ring-spot virus have been studied under electron microscope and found to consist of mainly virus particles (Rubio-Huertos and van Slogteren, 1956; Rubio-Huertos, 1956).

Intracellular inclusions seem to be associated only with rod

shaped virus particles. Microcrystals of tomato bushy stunt have been observed in the cells of infected *Datura stramonium* (Smith, 1956).

Kassanis (1939) and Bawden and Kassanis (1941) have shown crystalline inclusions in the nuclei of tobacco cells infected with severe etch virus and are mainly in the form of thin plates, which when viewed from the edge seem to be birefringent. Matsui and Yamaguchi (1964) studied the same and failed to detect any virus or virus like patterns within the inclusions.

Intranuclear crystalline inclusions have been observed in leaves of *Vicia faba* infected with bean yellow mosaic virus.

It is now more than proved that symptoms change with environmental conditions. The same symptoms are not caused by the same virus under all conditions. Virus-like symptoms could be produced by toxic substances, root damage, weather changes and deficiency of essential elements in the soil, e.g., potato leaf roll like symptoms could be caused by fumigation of soil with tetrachloroethane. Genetical abnormalities in plants also resemble effects produced by viruses. However, transmissibility distinguishes virus diseases from disorders of other origin.

Symptoms of plant viruses have been described in greater detail by Bos (1964).

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2

Transmission

The basic property of a virus is its ability to be transmitted to healthy plant species and cause the disease. The plant pathologist has to find ways and means of preventing its natural transmission. Since any virus is identified by the property of transmissibility, most of the research of the plant pathologist has been to discover different methods of transmitting viruses at will under controlled conditions of growth.

Five important methods are known for the transmission of plant viruses: grafting, mechanical, pollen, seed and vectors.

GRAFTING

Virus diseases which are systemic can be transmitted by grafting provided grafting is possible. As such, any virus can be transmitted by grafting. The only limiting factor is tissue compatibility of stock and scion. There are various grafting methods used in virological work.

Cleft or wedge grafting: This method is commonly used for herbaceous plants. The scion is trimmed on both the sides to a wedge shape and the stem of the stock is then cut across, preferably through the nodes and a vertical cut is given in the stem sufficient in length to accommodate the cut portion of the scion. The scion is inserted into the vertical slit and tied with a rubber or alkathene tape, waxed cloth, adhesive tape or sanhemp strands firmly. The plant is kept in humid atmosphere to allow the union to take place.

Bud grafting: The bud is inserted in a T-shaped opening made in the bark of the stock, and bound in place with a rubber or alkathene tape, but the bud itself is left exposed.

Approach or inarch grafting: Grafting is achieved by slicing of equal areas of cortical layers on one side of each of the two stems and binding the cut surfaces together with rubber or alkathene tape so that they remain in contact. The grafts are then moistened and are left exposed.

Patch or bark grafting: It consists of inserting a piece of infected bark tissue including cortex and phloem between the bark and inner tissues of healthy trees. It has been successfully used in case of sandal spike (Sreenivasaya, 1948).

Core grafting: In potatoes this type of graft is achieved by removing a core or cylinder by means of a cork borer containing an eye and inserting it into the hole made and core removed from the other to fit in the former.

MECHANICAL TRANSMISSION

Most of the basic knowledge of plant viruses has resulted from mechanical inoculation. The demonstration of infectivity (Mayer, 1886) of filterability (Iwanowaski, 1892), of acquired immunity (Wingard, 1928), of latency (Johnson, 1925), of local lesion assay (Holmes, 1929), of infectivity of virus crystals (Stanley, 1935), of infectivity of nucleic acid (Gierer and Schramm, 1956), of reconstitution (Fraenkel-Conrat 1959), all depend on mechanical transmission.

The earlier workers used a variety of inoculation methods. Mayer (1886) sucked juice of diseased plants into glass capillaries and inserted these into midribs of healthy plants. Beijerinck (1898) injected juice with a syringe and inserted pieces of dry leaf in wounds in the stem. Both Clinton (1915) and Allard (1918) mentioned that a high percentage of infection is obtained by lightly rubbing soybean and tobacco leaves, respectively, with diseased plant extract by hands or a brush. Schultz and Folsom (1923) inoculated potatoes by brushing leaves with their fingers and then applying juice of virus affected potato leaves. Fromme *et al.* (1927) and McKinney (1928) pointed out that the apparent efficiency can be achieved by nipping or swabbing leaves with juice or extracts of diseased plants.

The efficiency of the inoculum is greatly improved by the addition of an abrasive like carborundum powder (silicon carbide), 300 to 500 mesh or celite, sand or animal charcoal. The abrasive

causes slight injury of the cells and facilitates penetration of the juice. It removes the cuticle and hair and makes the leaf highly susceptible to infection. It increases the number of cells that are damaged during the rubbing of the juice on leaves. Yarwood (1953) suggested that use of phosphate buffer for extraction of juice increases the susceptibility by increasing the size and number of cell perforations. According to Hildebrand (1956) use of phosphate prevents harmful action of the host tissue sap on the virus and protects the wounds and the exposed protoplasm by filming over leaf surface. Some viruses are rapidly inactivated by components of sap particularly oxidising enzymes but in case of others, extracts from plants contain substances that inhibit infection, e.g., glycoproteins from *Phytolacca decandra*. Tannins are common components of several plants and their inhibitory effects can be overcome by grinding leaves in the presence of nicotine sulphate which combines with and precipitates the tannins. Raising the pH of leaf extracts by grinding in alkaline buffer solutions or by grinding leaves with solid disodium hydrogen phosphate also decreases the inhibitory action of tannins.

Needle Pricks

Some viruses are not easily transmitted by the rubbing method and require to be introduced by piercing an insect pin through a drop of infective juice by placing it in the axil of leaf, vein or petiole and covering the wound with a piece of sterilised cotton dipped in infective juice. Severin (1924) transmitted curly top of sugarbeet by needle punctures made through juice dropped into the heart of the healthy test plants.

SEED

Virus transmission through seed is more common in legumes and cucurbits. However, some important virus diseases are also transmitted through seeds of other crops like barley, lettuce, tomato, etc. For example, tobacco ring spot virus is seed borne to the extent of 50–80 per cent in soybean variety Lincoln, 20 per cent in petunia but not at all in tobacco (Henderson, 1931). Therefore, the ability to invade and survive in the seed is not an absolute property of a virus or virus strain but depends equally on the identity of the host plant. Cucumber mosaic virus is transmitted

by seeds of wild cucumber, *Micrampelis lobata* but not through the seeds of the cultivated variety (Doolittle and Gilbert, 1919).

It is believed that some viruses like TMV are not carried in the embryo, whereas bean mosaic virus infects the embryos either by pollen or by infecting the young embryo during early stages of its development. Crowley (1957a, b) and Singh *et al.* (1960) have reported that percentage of seed transmission in bean mosaic and barley stripe mosaic depended on the temperature at the flowering stage. Couch (1955) and Grogan *et al.* (1958) observed that variety of host plant can have great influence on the percentage of seed transmission of a virus in a plant. It seems likely, therefore, that any factor physical or genetical that can influence the concentration or survival of infectious virus particles in floral meristem of the plant will have effect on the seed transmission.

POLLEN

Blakeslee (1921) pointed out that the mosaic of *Datura stramonium* was transmitted through the pollen to the seed to seventy-nine per cent of the offspring. Reddick (1931) offered evidence that the bean mosaic virus is also transmitted by this method. In 1936, Reddick showed that potato virus Y entered the embryo from pollen but the percentage of seed transmission was found to be very low. Das and Milbrath (1961) infected squash plants with stone fruit ring spot virus through pollen.

VECTORS

Most plant viruses are transmitted from infected to healthy plants through an agency known as vector. It has been demonstrated that almost all types of organisms feeding upon or parasitising plants are capable of acting as vectors and these include biting and sucking insects, mites, beetles, nematode worms and chytrid fungi.

Aphids

According to Watson and Roberts (1940) and Sylvester (1956) aphid transmitted viruses can be divided into three groups, namely, non-persistent, semi-persistent and persistent based on the length of the period the infective or viruliferous aphids remain capable of transmitting the virus. Kennedy *et al.*, (1962) have introduced

the relationship between virus and aphids and gave a term stylet borne virus for non-persistent, and circulative and propagative for persistent aphid transmitted virus. A stylet borne virus can be picked up by the insect almost immediately after feeding and transmitted to susceptible healthy plant, all this can be accomplished in a minute or so. In serial transfers, in this kind of transmission, usually only the first plant is infected, and the insect then loses its infectivity. 'Circulative' viruses are retained by the aphid and have to pass through the gut wall into the blood and back to the salivary glands before the insect becomes infective.

Non-persistently transmitted viruses

Banana mosaic (Capoor and Varma, 1970); Barley mosaic (Dhanraj and Raychaudhuri, 1969); Bean mosaic (Yaraguntaiah and Nariani, 1963); Cardamom 'chirke' disease (Raychaudhuri and Chatterjee, 1961, 1965); Chilli mosaic (Nariani and Sastry, 1962) and Pea mosaic (Sreenivasan and Nariani, 1967).

Semi-persistently transmitted viruses

Cardamom 'Katte disease' (Uppal *et al.*, 1945; Varma and Capoor, 1958; Varma, 1962).

Persistently transmitted viruses

Barley yellow dwarf (Nagaich and Vashisth, 1963); Cardamom dwarf or foorkey disease (Varma and Capoor, 1964) and Groundnut chlorosis (Sharma, 1966).

Leaf hoppers

Next to aphids, leafhoppers are the most important vectors of virus diseases. Almost all the leafhopper borne viruses are transmitted in a persistent manner except rice tungro virus which is transmitted in a non-persistent manner by *Nephotettix impicticeps* (Raychaudhuri, *et al.*, 1966; John, 1968). Capoor *et al.*, (1968) reported transmission of chlorosis of jowar by *Peregrinus maidis* in India. Other examples of leafhopper transmitted viruses are maize streak by *Cicadulina mbila*, rice dwarf by *Nephotettix nigripictus* and beet curly top by *Circulifer tenellus*. The main features of the leaf hopper transmission are (i) ability to transmit is not increased by fasting, (ii) inability to transmit immediately after acquisition of the virus or presence of incubation or latent period, (iii) retention of the infective power for long periods. Several of the viruses transmitted by leafhoppers have been shown to multiply in their vectors. Leafhoppers are also known to be the vectors

of yellows type of diseases attributed to mycoplasmas, e.g., *Hishimonas phycitis* vector of brinjal little leaf, *Macrosteles fascifrons* vector of Aster yellows, *Dalbulus maidis* vector of corn stunt.

Thrips

Bald and Samuel (1931) described the transmission of tomato spotted wilt virus by *Frankliniella insularis*. Other species of thrips have also been found to be vectors. Several days elapse between the vector acquiring the virus and becoming infective. Once the latent period is over the insects remain infective for the entire life.

Insects must feed during their larval stages on an infected plant to become infective. Both larvae and adults can transmit, but larvae that do not feed on an infected plant till they are adults do not transmit however long they live. There is no correct explanation for this, but transmission obviously involves the virus circulating through the vector's body and may involve virus multiplication and one possibility is that some tissues change and lose permeability to the virus as the insect becomes adult.

White flies

All the white-fly transmitted viruses seem to behave in much the same way in their vectors with a latent period from a few hours to a day or more between acquiring virus and becoming able to transmit. White fly can acquire virus in acquisition feeding of thirty minutes, but the proportion of insects that becomes infective increases with increasing time on infected plants. Similarly, although fully infective white flies can infect healthy plants in feeding periods of only ten minutes, most require longer period (Costa and Bennett, 1950; Varma, 1955). Other viruses which are transmitted by white flies are leaf curl of tobacco (Pruthi and Samuel, 1937); yellow mosaic of *Acalypha indica* (Chenulu and Phatak, 1965), yellow mosaic of *Phaseolus aureus* (Nariani, 1960).

Mathur (1932) reported the occurrence and transmission of zinnia leaf curl by *Bemisia tabaci*. These viruses are obviously contained in and circulate through the bodies of their vectors.

Mealy bugs

Mealy bugs are much more sedentary than other vectors of viruses. Viruses like swollen shoot rarely spread between cacao trees

unless these are in contact. This spread is probably by crawling nymphs of mealy bugs, which are not only more mobile than adults but also more efficient vectors, or by mealy bugs moved from tree to tree by the ants which tend them. The occasional spread over long distance presumably happens because mealy bugs are borne by the wind or because infected and infested cacao pods and other parts of trees are carried over long distances.

The virus content of infected plants is small, few infections are obtained when cotyledons are infested with single mealy bug from infected plants. Increasing the number of mealy bugs increases the proportion that become infected and 95 per cent may become infected with twenty-five insects per cacao bean, in case of swollen shoot of cacao.

Mites

Fig (*Ficus palmata*) mosaic (FMV) and pigeon pea (*Cajanus cajan*) sterility (PSV) are transmitted by eriophyid mites in India. I MV is transmitted by *Aceria ficus* occurring in nature on fig plants (Vashisth and Nagaich, 1968) and PSV by *Aceria cajani* found only on pigeon pea (Seth, 1962, 1965).

For transmitting the viruses healthy mites are allowed to feed on diseased plants for four days (Seth, 1962) or a week (Vashisth and Nagaich, 1968).

Beetles

Since the beetles have no functional salivary glands, it is necessary for them to regurgitate part of the contents of the foregut while eating, this is apparently essential to help the digestion process. Regurgitation brings into contact with the leaf the infective tissue previously eaten and this, during mastication is inoculated to a healthy susceptible plant. Viruses like squash mosaic (Freitag, 1941), turnip yellow mosaic (Markham and Smith, 1949), turnip crinkle and turnip rosette (Broadbent, 1957), and potato virus X (Nagaich *et al.*, 1972) are found to be transmitted by beetles.

Grasshoppers

Walters (1952) showed that the large grasshoppers, *Melanoplus differentialis* could carry the virus, tobacco mosaic, in its jaws and infect a healthy tobacco plant after first feeding on a mosaic infected plant. It has been found that the saliva plays an

important role in the transmission of viruses by insects.

The turnip yellow mosaic virus is transmitted by two species of grasshoppers and an earwig but not by caterpillars (*Pieris* sp.) or any sucking insects (Markham and Smith, 1949) and turnip crinkle virus by hoppers of *Locusta migratoria* and leaf-mining larvae of the fly *Phytomyza rufipes* (Martini, 1958).

Nematodes

There are ten orders of phylum Nematoda (Goodey, 1963). Most of the parasitic nematodes on living green plants belong to the order Tylenchida, but none of this group has yet been found to be a virus vector. Vectors known so far are confined to the order Dorylaimida group containing only a few parasites, in the parasitic genera, *Xiphenema*, *Longidorus* and *Trichodorus*. *Xiphenema index* transmits fan leaf virus of grapes (Hewitt *et al.*, 1958) and grapevine yellow mosaic virus (Vanek *et al.*, 1972). Other examples of transmission of plant viruses by nematode vectors are arabis mosaic by *X. diversicaudatum* (Jha and Posnette, 1961), tomato black ring by *Longidorus elongatus* and *L. attenuatus* (Harrison *et al.*, 1961) and tobacco rattle by *Trichodorus pachydermus* (Sol and Seinhorst, 1961).

Fungi

Some of the soil borne viruses have recently been shown to be transmitted by fungi. Tobacco necrosis virus (TNV) forms an interesting group of biologically similar but serologically unrelated soil borne viruses (Bawden and Pirie, 1942) and until recently there has been much speculation on their mode of transmission. It has been now conclusively shown that these viruses are transmitted by *Olpidium brassicae* (Teakle, 1962).

Some recent work by Fry and Campbell (1966) gives details of the relationships between the fungus, the virus, and the host. When suspensions of *Olpidium* and tobacco necrosis virus (TNV) were filtered so as to remove all spores, the filtrate which still contained infective TNV was non-infective to the plant. Plants became infected only when exposed to mixtures of TNV and zoospores.

Other examples of transmission of plant viruses by fungi are lettuce big vein and tobacco stunt by *Olpidium brassicae* (Fry, 1958; Grogan *et al.*, 1958; Hidaka, 1956), wheat mosaic by *Polymyxa graminis* (Estes and Brakke, 1966), potato virus X by

Synchytrium endobioticum (Nienhaus and Stille, 1965) and potato mop top by *Spongospora subterranea* (Jones and Harrison, 1969).

Dodder transmission

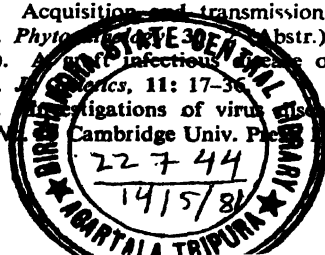
Dodder (*Cuscuta* spp.) is a parasite on higher plants and lacks leaves and chlorophyll. The parasite forms haustoria, which are organs penetrating the vascular tissues of the host. Bennett (1940) observed that dodder would transmit viruses from plant to plant, e.g., sugarbeet curly top virus (BCTV) with *C. subinclusa* and CMV with *C. californica*. Johnson (1941) transmitted aster yellows, CMV, BCTV, tomato bushy stunt virus (TBSV) and TMV with *C. campestris*.

Dodder can be used to transmit a virus between distantly related plants. The virus being transmitted experimentally may not multiply in the dodder. The dodder then seems to act as a passive pipeline connecting two plants, e.g., TMV does not seem to multiply in the dodder. Effective transmission is obtained only if the same dodder plants connect the diseased and healthy plants (Cochran, 1946). In some respects this is similar to grafting.

Kunkel (1943, 1945) transmitted cranberry false blossom virus from cranberry to twenty-eight different species belonging to ten different families including convenient plants like tobacco and tomato. Peach rosette has been transmitted to tobacco, tomato and other plants.

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3

Physiology of Infected Plants

The most common physiological and biochemical changes brought about by viruses in plants are (i) an increase in respiratory rate, (ii) increase in the activity of certain enzymes, particularly polyphenol-oxidases and accumulation of oxidised polyphenols, (iii) a decrease in the rate of photosynthesis, (iv) decreased activity of plant growth regulators. The early workers mostly presented a comparative analysis of healthy and virus infected tissues with regard to total carbohydrates and sugars; total nitrogen, carbon/nitrogen ratio, rates of photosynthesis and respiration and the components of ash. In the recent years, however, detailed work on possible mechanisms by which virus infection may affect these processes has been attempted.

Some workers consider that mature chloroplasts are destroyed in mosaic affected leaves while others believe that no destruction of chlorophyll takes place but the chloroplast formation is inhibited. Both views may be correct depending upon the virus concerned. In aucuba mosaic of tomato, the virus does not affect the chloroplasts of the leaves which are fully developed at the time of infection but it prevents the formation of plastids in the young growing leaves. On the other hand, certain viruses like tomato stripe and cucumber mosaic produce chlorosis in inoculated leaves. Esau (1933) also described destruction of chloroplasts in the leaves of beet plants infected with curly top virus. Cook (1926) investigated the chlorophyll content of mosaic affected sugarcane and concluded that the development of chloroplasts were retarded rather than inhibited or destroyed. Peterson (1931) and Peterson and McKinney (1938) studied the influence of mosaic diseases on plastid pigments and chlorophyllase in tobacco leaves and noticed that the drop in

chlorophyll content was accompanied by an approximately proportional drop in the yellow pigments, carotene and xanthophyll and chlorophyllase activity was more in chlorotic tissues.

Increased enzyme activity has been recorded as a result of infection with many viruses such as TMV (Woods, 1899, 1900; Solymosy and Farkas, 1963), beet curly top (Bunzel, 1913), tomato spotted wilt (Best, 1937) and potato leaf roll (Rouzinoff, 1930). Reddy (1963) found enhanced peroxidase activity and reduced catalase activity in cowpea plants infected with mosaic virus in India. It has been suggested that the increased polyphenol oxidase activity found in necrotic local lesions caused by viruses like TMV is directly concerned in limiting the spread of virus in necrotic lesions (Farkas *et al.*, 1960; John and Weintraub, 1967). Solymosy *et al.* (1967) examined the peroxidase activity in *Phaseolus vulgaris* and *Nicotiana glutinosa* following infection with different viruses and found that the peroxidase enzymes formed after virus infection are specified by the host rather than the virus.

Accumulation of starch in the leaves is particularly marked in such diseases as the potato leaf roll, spinach blight, peach yellows and little peach. In potato leaf roll, in addition to accumulation of starch in the leaves, there occurs necrosis of the phloem. Quanjer (1913) considered this accumulation of starch to be due to phloem necrosis which prevented transport of carbohydrates. But Murphy (1923) found that rolling of leaves and accumulation of starch could occur in the absence of phloem necrosis. Barton and McBain (1932) made a careful comparison of the carbohydrate metabolism of normal with that of leaf roll affected potatoes and discovered that in healthy plants hexose was the first sugar of photosynthesis not sucrose and that sucrose was the sugar of translocation. In leaf roll affected plants photosynthesis is reduced and the main reactions were conversion of starch to hexose, hexose to sucrose and sucrose back to starch. Thus hexose is sugar of translocation in diseased plants and sucrose is absent and plays no part in translocation. Investigations with several viruses have shown a reduction in photosynthetic activity in infected leaves which usually begins some days after infection (Owen, 1957a, 1958, Roberts and Corbett, 1965; Jenson, 1968a; Tu and Ford, 1968; Tu *et al.*, 1968). Studies on photosynthesis of sugarcane mosaic plant showed (Cook, 1926) that the healthy plants and green areas of diseased plant show a large amount of starch in the

afternoon and very little in the morning while the chlorotic areas show a small amount of starch in the afternoon and practically none in the morning. This shows that the starch forming power of the mosaic affected cane is reduced in proportion to the amount of infection while the power of translocation is almost unimpaired.

Earlier work of Dunlap (1930) suggested that the mosaic diseases are accompanied by an increase in the total nitrogen and a decrease in the total carbohydrate content of the foliage resulting in decreased carbon/nitrogen ratio while in case of yellows diseases the reverse is true. The later work (Takahashi, 1941; Bawden and Kleczkowski, 1957) has however, shown that no consistent pattern is evident for the effect of virus infection on soluble proteins and there was no correlation between the content of normal proteins and either severity of symptoms or virus content of the leaves.

Dunlap (1930) reported an increase in the respiration in young tissues affected with virus diseases, while in older diseased leaves respiration was less than in healthy tissues. Increased respiration due to virus infection has been reported for several viruses such as potato leaf roll (Thung, 1928; Whitehead, 1931), brome grass mosaic (Burroughs *et al.*, 1966), dolichos enation mosaic (Ramadasan, 1962), cowpea mosaic (Reddy, 1966) maize dwarf mosaic (Tu and Ford, 1968), barley yellow dwarf (Jenson, 1968a, b), etc. Caldwell (1934) found that tomato plants inoculated with aucuba mosaic at five leaf stage evolved more carbon dioxide than healthy controls but when inoculated at seedling stage showed lower output in the beginning followed by higher output than the healthy plants. On the other hand, Lemmon (1935) and Takahashi (1947) found that the respiration rate of healthy plants was always higher than that of diseased. Kempner (1936) was unable to find any change in the respiration of mosaic infected tobacco leaves. Glasstone (1942) found that the respiration rates of healthy and mosaic infected plants began at approximately equal levels and remained equal upto a point at which the respiration rates of the diseased plants rose suddenly above the rate of the healthy plants. Then in a few days this high rate of respiration in the diseased plants gradually decreased until the rates were approximately equal again in diseased and healthy plants. Ramadasan (1962) reported that the rate of respiration of *Dolichos lablab* leaves infected with dolichos enation mosaic virus increased and was maximum on

fourth day after inoculation but by the eighth day it decreased and was even lower than healthy leaves. Owen (1955a, b, 1956, 1957a, b, 1958) reported variable effects in respiration rates depending on several factors such as age of leaves, time of inoculation, environmental conditions and the virus involved. Owen (1955a, 1956) observed that the respiratory rates differed in plants grown in different times of the year. During winter the infection increased respiration rates and in summer decreased them. Owen (1957a, b) also showed that different viruses may affect the respiration of the same plants in different ways. Unlike tobacco mosaic virus which increased the respiration of tobacco leaves within an hour of being inoculated, the tobacco severe etch virus did not change the respiration rate until the leaves showed external symptoms. The respiration rate of inoculated or systemically infected leaves with symptoms rose to 40 per cent above that of healthy leaves, three times the increase produced by tobacco mosaic virus.

With host-virus combinations resulting in necrotic lesions, there are consistent reports that a rise in respiration rate accompanies the appearance of lesions, Weintraub *et al.* (1960) and Sunderland and Merrett (1965), found that the rise in respiration rate occurred several hours before necrosis became visible. Farkas and Solyomosy (1962) and Solyomosy and Farkas (1963) observed that enzymes of pentose phosphate pathway were increased in the zone of yellow tissue surrounding developing necrotic lesions due to TMV. Bell (1964) reported stimulated respiration and increased activity of pentose phosphate pathway in other host-virus combinations producing local lesions such as alfalfa mosaic and southern bean mosaic viruses on some varieties of *Phaseolus vulgaris*, whereas Merrett and Sunderland (1967) found the increased respiration in xanthi tobacco leaves showing necrotic local lesions involved both the glycolytic and pentose phosphate pathways.

Virus infection could influence plant growth by increasing or decreasing the synthesis, translocation or effectiveness of growth regulating substances in different organs. The findings of many workers using different viruses and host plants agree in suggesting that virus infection reduced the auxin activity in leaf extracts, e.g., in case of tomato spotted wilt in tomato (Grieve, 1943), TMV in tomato (Pavillard, 1952) and tobacco (Pavillard and Beauchamp, 1957) and sugarbeet curly top virus in various hosts (Smith *et al.*,

1968), Application of gibberellic acid has been reported to reverse the virus induced stunting in case of tobacco etch virus in tobacco (Chessin, 1957) wound tumor virus in clover (Maramorosch, 1957) and tobacco leaf curl virus in tomato (Lal and Singh, 1961) and tobacco (Nariani, 1963), suggesting the possibility that viruses might affect the auxin concentration of the plants. Ross and Williamson (1951) detected ethylene gas (another growth regulator) in leaves from *Physalis floridana* plants infected with potato virus Y in appreciable amounts. Since symptoms produced by potato virus Y in *P. floridana* resemble those produced by plants exposed to ethylene gas, this led them to believe that production of ethylene gas by the virus was responsible for these symptoms.

Very little work has been done on the possible effects of virus infection on host RNA, ribosomes and mineral elements and it is not possible to draw definite conclusions.

Porter and Weinstein (1960) found no significant difference in DNA content between healthy tobacco plant and plants inoculated with cucumber mosaic virus over a period of 280 hours after infection. On the other hand, Misawa *et al.* (1966) found that there was a transitory increase in the content of DNA in nuclei in tobacco leaves four to ten hours after inoculation. Honeycutt and Millikan (1964) reported that *Prunus mahaleb* leaves doubly infected with certain viruses and sampled several months after infection showed a reduction in DNA content.

Shigematsu *et al.* (1966) observed that RNA present in tobacco leaves forty-eight hours after inoculation with TMV increased about four fold but Kubo *et al.* (1965) using similar methods found no such increase. Matthews (1958) also reported no gross change in overall RNA content in turnip yellow mosaic virus (TYMV) infected chinese cabbage leaves. Babos (1966a) on the other hand reported that the activity of RNA isolated from a nuclear chloroplast fraction and from ribosomes was the same for healthy and TMV infected tissues. Between 80 to 140 minutes the activity of RNA in infected leaves was higher than in control. Semal and Kummert (1967) found that the overall RNA synthesis increased by about 50 per cent in young systemically infected leaves of barley with brome grass mosaic virus but no difference was observed in inoculated old leaves.

Reddi (1963) suggested that there is a rapid breakdown of host ribosomes following TMV infection and the nucleotides from

ribosomal RNA are used for viral RNA synthesis. Hirai and Wildman (1963), found that during early systemic infection by TMV in tobacco, chloroplast ribosome and protein synthesis was inhibited, while cytoplasmic ribosome synthesis was not. Israel and Ross (1967) observed a marked increase in the amount of endoplasmic reticulum and associated ribosomes in mesophyll cells surrounding necrotic local lesions induced by TMV in *Nicotiana glutinosa*. On the other hand, Babos (1966b) found no difference in ribosome content in healthy and TMV infected tobacco leaves.

Viruses are known to bring about a change in amino acid content of infected plants (Allison, 1953; Henke, 1957; Porter and Weinstein, 1960; Selman *et al.*, 1961; Bozarth and Diener, 1963; Narayana Swamy and Ramakrishnan, 1966; Welkie *et al.*, 1967). No consistent results are, however, available to suggest a particular trend in increase or decrease in amino acid content applicable to a large number of viruses.

Studies conducted on transpiration rate and water content of virus infected plants suggest that these are lower in diseased plants in well established infections (Pantanelli, 1952; Gondo, 1953; Owen, 1958; Orlob and Arny, 1961).

It has been reported that there is a marked accumulation of scopoletin around the primary lesions induced by tomato spotted wilt virus and around lesions induced by TMV in *N. glutinosa* (Best, 1936, 1944). It has been suggested that scopoletin may accumulate at the expense of indoleacetic acid (Pavillard and Beauchamp, 1957). Increases in the concentration of certain other phenolic compounds has also been reported following virus infection (Geismann, 1956; Hampton *et al.*, 1964; Gill, 1965). The accumulation of most of these aromatic compounds has been suggested to be non-specific connected with death of cells due to injuries caused by pathogenic organisms or other agents.

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4

Purification of Plant Viruses

The object of virus purification is to separate the virus nucleoprotein from other components of the host cell and to obtain it in a biologically active state in the form of a concentrated monodisperse suspension.

Several steps are involved in virus purification, namely, extraction, removal of all contaminants and concentration.

Extraction can be achieved by homogenisation. It can be done by using a mortar with a pestle, a waring blender, or if large quantities of leaves are used and losses are irrelevant in an ordinary meat grinder. The homogenate is then pressed through cheese cloth to remove large cellular debris.

Deep freezing of the leaves before homogenisation often gives higher virus yields. This can be performed only with viruses whose infectivity is not destroyed by freezing. Viruses can be protected from inactivation by extracting them from plant tissue in the presence of a buffer or in the presence of reducing agents, such as ascorbic acid and cysteine. Clarification of the sap is applied for the removal of most of the cell components other than the virus nucleoprotein. It is carried out by heating and freezing of the sap or by treating the sap with organic solvents like butanol and chloroform, ethanol, acetone, etc., or simply by filtration with filter aids such as celite, bentonite, charcoal, etc., or by low speed centrifugation. At the end of this process the virus is in the supernatant

Concentration can be carried out by two methods; chemical and physical.

Chemical methods include salting out (usually with ammonium sulphate) and by precipitation (alcohol, acetone, isoelectric point).

Any of these steps is followed by centrifugation. Now the virus particles are in the sediment and can be resuspended in an appropriate buffer.

Physical methods have recently been used to an increasing extent because they are more gentle than chemical methods, and therefore, can be used for the purification of labile viruses without any appreciable loss of infectivity (Black, 1955).

Plant viruses are separated on the basis of their size by ultracentrifugation and molecular sieving techniques and on the basis of their surface charges by electrophoresis and ion-exchange chromatography.

For many viruses repeated low and high speed centrifugation leads finally to a virus preparation of reasonable purity. One method of further separating of virus particles from other high molecular weight contaminants by ultracentrifugation is density gradient centrifugation (Brakke, 1960) in sucrose or in cesium chloride. Gradient columns of sucrose solution are made by layering sucrose solutions of different concentrations in centrifuge tubes in such a way that the highest concentration is placed at the bottom of the tube, then the next lower concentration and so on, the lowest concentration being on the top. The virus suspension is placed on the top of this column and the tubes centrifuged in a swinging bucket rotor at fairly high speeds ranging from 20,000 to 40,000 rpm. Under centrifugal force the particles in the gradient sediment according to their size, shape and density, reach a position where density is equal to that of the medium and form a zone or band. The bands may be visualised due to their light scattering in dark by throwing a narrow beam of light from above and can be removed using a hypodermic syringe or by puncturing the bottom of the tube and collected into test tubes.

The rate zonal centrifugation method is that in which the centrifuge is stopped before the various components have reached their own density level, but are still separated to an acceptable extent from contaminants.

In both the above cases the tube is punctured at the bottom with a hypodermic needle and the layer containing the virus particles is drained.

Another useful physical method for the separation of virus particles on the basis of size and shape is molecular sieving through columns of chopped or bead form agar or agarose gel (Steere and

Ackers, 1962). A long narrow column of Sephadex, agar or agarose is prepared containing the gel grains equilibrated with a suitable medium or buffer. The virus extract in buffer solution is applied to the top of the column and continuously fed with buffer solution. Virus particles while passing through the spaces between the granules will flow at faster rate than other low molecular weight materials. The first fractions collected at the bottom are usually free from contaminants and can be tested by light scattering, infectivity and serological activity for the presence of virus.

Ion-exchange chromatography has successfully been applied by Shainoff and Lauffer (1957). For the purification of plant viruses, electrophoresis in agar gel columns (Zone electrophoresis) has been worked out by Townsley (1959), e.g., TMV. Townsley (1959) was able to separate TMV from potato virus X by using this method.

Some plant viruses can be crystallised. Salting out is one of the best methods to get plant viruses in a crystalline state (tomato bushy stunt virus, southern bean mosaic virus, TMV). When purified tobacco ringspot virus is stored at 4°C in phosphate buffer, it crystallises (Steere, 1956). Turnip yellow mosaic virus (Markham and Smith, 1949) as well as tobacco necrosis virus (Bawden and Pirie, 1945) have been obtained in a crystalline form at the end of the purification procedure.

The homogeneity of the purified virus preparation may be checked by spectrophotometry, electrophoresis, ultracentrifugation and electron microscopy. The infectivity per unit volume of the preparation should be compared to that of the original sample. This kind of comparison will show the specific infectivity of the preparation.

Stanley and Anderson (1941) purified five different viruses including cucumber viruses 3 and 4 and tobacco mosaic virus by differential centrifugation and studied them under the microscope. Both revealed close similarities with respect to size (length of 300 nm) and shape. Takahashi (1948) conducted electron microscopic study of the purified squash mosaic virus and reported the virus particles to be spherical with an average diameter of 30 nm. The virus was also obtained in a crystalline form, the crystals measuring $0.5-0.8 \times 3-5 \mu$.

Rice *et al.* (1955) purified squash mosaic virus from sugar pumpkin by a combination of chemical precipitation and

differential centrifugation which gave three components on density-gradient centrifugation but in the freeze dried electron micrograph preparation containing all the three components (111 S, 88 S and 56 S) the most predominant particles appeared as polyhedrons with a diameter of 33 nm.

Inoeye *et al.* (1967) reported cucumber green mottle mosaic virus from Japan. The virus particles were straight rods 300×18 nm in size. van Regenmortel (1969) purified a watermelon mosaic virus from South Africa using chloroform and differential centrifugation and resuspended the pellet in sugar solution in borate buffer. The purified preparation contained virus particles 700–800 nm, long and 13 nm wide. Continuing his studies on a ringspot virus isolated from squash van Regenmortel (1969) partially purified the virus by chemical means, differential centrifugation and gradient zone electrophoresis. The virus had a sedimentation constant of 104 S, compared with 98 S determined by analytical centrifugation and calculated particle size of 26 nm in diameter.

Using the method of purification earlier employed from watermelon mosaic virus, van Regenmortel (1961) purified a cucumber mosaic virus isolate from lupines and subjected to zone electrophoresis. The purified virus preparations before zone electrophoresis gave a mean diameter of virus particles as 48 nm and that after electrophoresis, 30 ± 1 nm.

In India virus purification work started with report of purification and crystallisation of sunn hemp mosaic virus (SMV) by Raychaudhuri (1947).

Capoor (1950) reported purification of datura distortion mosaic virus (DDMV). Anand (1960) described a method on purification of bottlegourd mosaic virus from bottlegourd. The electron micrographs revealed the virus to be flexuous rods of variable length 100–1350 nm and 25 nm in width.

Phatak and Verma (1967) purified a strain of TMV from potato. The purified virus revealed rigid rods 230 nm long and 13 nm wide. Anand (1968) reported the successful purification of another virus from sunn hemp (*Crotolaria juncea*) designated as southern sunn hemp mosaic virus. This was different from the SMV of Raychaudhuri (1947) because the particles are rigid rods resembling those of TMV. Nariani *et al.* (1970) purified the southern sunn hemp mosaic virus using butanol and differential

centrifugation. The virus had a modal length of 300 nm and average diameter of 18 nm and was found to be serologically related to TMV.

Chenulu *et al.* (1968) purified an isolate of cowpea mosaic virus. Electron micrographs revealed the virus to be spherical with diameter of 23 nm. Chenulu *et al.* (1968) purified a virus causing a mosaic disease of henbane (*Hyoscyamus niger*) by the agar gel filtration method. The virus as seen under the electron microscope was a less rigid rod of variable length 270 to 750 nm.

Skanker *et al.* (1969, 1971, 1972) reported successful purification of common cucumber mosaic virus from snakegourd (*Trichosanthes anguina*), bottlegourd mosaic virus from bottlegourd, watermelon mosaic virus from watermelon and pumpkin mosaic virus from pumpkin by means of differential centrifugation and several other methods including density gradient centrifugation. The purified cucumber mosaic virus when seen under electron microscope revealed typical spherical particles 29 nm across. Bottlegourd mosaic virus and watermelon mosaic virus were observed as rod shaped particles with modal lengths of 280 nm and 240 nm, respectively. A fourth virus causing pumpkin mosaic was also purified by differential centrifugation and agar gel filtration and found to consist of long flexuous rods 840 nm in length.

Raychaudhuri *et al.* (1969) purified rice tungro virus (RTV) using the differential centrifugation method. The purified virus was found to be a sphere with a diameter of 33 nm. Employing a similar procedure they also purified a virus causing mosaic streak of wheat. The viral particles are spherical in shape with an average diameter of 40 nm.

Dhanraj and Raychaudhuri (1969) reported purification of barley mosaic virus from barley by differential centrifugation. The virus is spherical in shape with a diameter of 40 nm.

Summanwar *et al.* (1969) obtained purified preparations of a virus associated with coconut root (wilt) disease by differential centrifugation of sap from infected coconut leaves and roots. The purified preparation when examined under the electron microscope showed typical rod shaped particles 320-360 nm long.

Subbayya and Raychaudhuri (1970) purified ragi mosaic virus by differential centrifugation. Electron micrographs revealed the particles to be flexuous rods with an average length of 667 ± 8 nm and an approximate diameter of 12-14 nm.

Chowfla and Nariani (1974) reported the purification of broad bean mosaic virus (BBMV) occurring in Delhi using 6 per cent butanol and differential centrifugation. The virus particles were found to be spherical with an average diameter of 25 nm.

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5

Serology

Serology is an indispensable tool in modern virus studies in unravelling group interrelationships, identity of particulate structure and diagnosis of the disease. Before describing in detail the serology of plant viruses, it will be better to understand some of the terms used.

When an animal is infected with a pathogen, such as a bacterium or virus, one response to infection is the appearance in the blood stream, of proteins which can combine specifically with the bacterium or virus. Such proteins are known as antibodies. Any substance which stimulates the production of antibodies and which combines with them *in vitro* is called an antigen. Generally antigens are proteins in nature. A few complex lipids, carbohydrates and poly-saccharides have been shown to be capable of producing antibodies, but the majority of antigens contain protein of relatively high molecular weight. A serum containing antibodies is called antiserum, while serum from an animal which has not been injected with any antigen is called normal. Induction of specific antibodies in an immunized animal can be enhanced by the use of adjuvants to obtain high titre antiserum.

Several widely differing animals such as sheep, pigs, chicken, frogs, horses and rabbits have been employed for antiserum production. The injections are intraperitoneal, intravenous, intramuscular, subcutaneous, or foot-pad (rabbits). The quantity of virus (antigen) used at each injection is about 0.01 ml to 2 ml.

It is believed generally that an antigen accumulates in the cells of the reticular endothelial system of an animal. The chief producers of antibodies are the immature plasma cells in the

reticular system. The intensity of production at any site may bear little relation to the concentration of reticuloendothelial tissues.

SEROLOGICAL METHODS

Serological methods were first applied to plant viruses by Dvorak (1927). And this process was systematically employed in plant taxonomy by Metz and his associates during the early nineteenth century. The specificity of the serological reactions of plant viruses was first established by Beale (1928). Beale (1937) has shown that mosaic affected tobacco plants contain an antigen specific for virus containing extract which is not present in the sap of healthy plants. It was shown by Gratia (1933a, b) that plants containing different viruses also contained different specific antigens; while Birkland (1934) was the first investigator to demonstrate that strains of plant viruses contained specific antigens which differentiated them from other members of the group.

Several types of serological methods have been used in plant virus work.

Precipitation Reaction

A precipitate is formed when the virus is added to its specific antiserum in saline at different dilutions and kept at 37°C in a water bath. In precipitation test the antibody is referred to as precipitin.

Complement Fixation Test

When antigens are mixed with their specific antibodies, the mixture has the property of removing the power of normal serum to haemolyse sensitised red corpuscles. It is a kind of decline colour indicator test. Complement is a thermolabile non-specific constituent of normal serum (Bawden, 1950; Matthews, 1957; Moorehead, 1956 and 1961; Wright and Hardy 1961; Wright, 1963). When antigens and antibodies unite, complement is either used up or fixed up. The amount fixed varies with the extent to which antigen and antibody react and is measured by observing whether or not there is still sufficient free complement to allow the lysis of the sheep blood corpuscles in the presence of anti-sheep-cell rabbit serum. This serum, known as the haemolytic amboceptor, is freed from its complement by heating to 56°C,

Fixation experiments are carried out in two parts. First, the antigen and antibody under test are mixed and complement added. After an hour, the washed sheep corpuscles and the haemolytic amboceptor are added and the mixture kept at 37°C. Readings are taken at intervals on the degree of lysis of the blood cells. The reactions taking place are given below:

1. Haemolytic amboceptor + Complement - Corpuscles = Lysis
2. Antigen + Antibody + Complement = Fixation
3. 2 - Haemolytic amboceptor - Corpuscles = No Lysis

Anaphylaxis

In this test the union between antigen and antibody is detected by reaction in animal tissues (Seastone *et al*, 1937; Beale and Siegel, 1941). In living animal, there may be violent spasmodic muscular contractions, inflammation of tissues or other abnormal effects, sometimes resulting in death. *In vitro*, the most sensitive form of anaphylactic shock is the Schultz-Dale technique (Dale, 1931). Virgin guinea pigs are immunised by injecting the antigen. After three weeks, they are killed and the two horns of the uterus are removed. To each of the horns is attached a thread. The uterine horn is then placed in an aerated Ringer's solution, kept at 37° C. The lower end is tied rigidly to the bath whereas the upper end is attached to a kymograph needle. A small quantity of the antigen is introduced into the Ringer's solution, and a positive reaction is shown by a rapid contraction of the uterine horn followed by a slow relaxation, both reactions being recorded on the Kymograph.

Neutralisation of the Infective Property of Virus

The self explanatory term implies that when viruses are mixed with antisera prepared against them, they lose their infectivity.

Haemagglutination

Saito and Iwata (1964) purified barley stripe mosaic virus by treating blood cells with tannic acid. Blood cells with tannic acid

treatment non-specifically absorb protein antigens when these cells with antigen attached to their surface can be incubated with antiserum. Specific combination between the antigen and the antibody will result in agglutination of the red cells. This method is found to be specific and highly sensitive. Because of the difficulty in getting repeatable tanning of cells, this method has not yet been widely used with other viruses.

There is another form of halmaggeutination used by Nelson and Day (1964) for cauliflower mosaic virus. Here the virus is made to bind with a complement before it reacts with antibody. The complex then adheres to appropriate red blood cells, causing them to clump.

Agglutination Test

When a drop of leaf sap, freshly obtained from a virus infected plant is mixed on microscopic slide with a drop of antiserum, clumping of small particles of host material occurs. This can be seen with the naked eye but is clearly viewed under a low powered microscope. Chloroplasts and chloroplast-fragments are prominent in the clumped aggregates.

Double Gel Diffusion

When antigen and antiserum are placed in a tube so that both reactants are permitted to diffuse into a blank column of agar, precipitation occurs. The number, density and position of each zone is characteristic of the antigen antibody system under investigation, but also depends upon some properties of the medium such as concentration of the agar and influence of various ions (Moorehead, 1961).

Microprecipitin Test

This test is designed to determine the amount of virus (antigen) or of antibody in very small amounts of materials. The test is performed under mineral oil, thereby permitting a large incubation period which would not be possible without oil. Plant extract, clarified by some appropriate chemical or physical method, as well as purified virus solution can be titrated by this method.

The petridish bottom is coated with Formvar and a check-board pattern is drawn with a wax pencil. Appropriate series of dilutions of antigen and of antiserum are made in test tubes

using saline as the diluent. Then a small drop of the appropriate dilution of antigen and antiserum is added to each square in the dish which is later flooded with mineral oil making each drop on the dish completely covered by the oil. A small drop of saline in each series serves as control. The dishes are then incubated and the reactions are read under a binocular dissection microscope.

Ring Interface Precipitin Test

Serum is placed in the bottom of small tubes, and antigen is carefully layered onto the surface so as to keep mixing at a minimum. Diffusion of the two solutions takes place upon incubation, forming a ring of precipitation where optimum concentrations of antigen and of antibody are present. The test is very sensitive in being able to detect small amounts of antigen (virus). However, the proper controls are of utmost importance since both serum and plant extract may produce spontaneous precipitation.

Ouchterlony Agar Double-Diffusion Test

When antigen and antibody are added to wells within an agar medium they diffuse into the agar at a given rate which is dependent on the density, concentration and composition of the agar as well as that of the reactants. Where antigen and antibody meet in optimum proportions in the agar, precipitation zones occur; the advantage of this test is that a number of dilutions of a given antigen or antiserum sample or a number of different samples can be tested at one time under identical conditions (van Slogteren, 1955).

Immunoelectrophoresis

This technique is based on the electrophoretic migration of a protein in a convection preventing medium (starch cellulose acetate or agar) and on the subsequent location of the protein components by precipitation with antiserum. Antigens and antiserum diffuse towards each other and precipitate where they meet in optimum proportions. The laws that apply to the tube or plate methods of diffusion also apply to immunoelectrophoresis.

Application of Serological Methods

The precipitation reaction has found extensive use with several adaptation and modifications for the diagnosis of plant viruses

both in the field and in the laboratory. Many practical applications of this method could be recognised but three important ones are as follows:

Identification of virus: The rapid agglutination test is valuable in identifying potato virus X, Y and S in the fields. This method has become a routine testing in several countries in the potato seed certification programmes

The slide agglutination test, suitable for routine rapid recognition of viruses, was first applied by Chester (1937) and further in Holland by van Slogteren (1955b). It involves the use of drop of crude infective plant sap which is mixed with the specific antiserum on a microscope slide. In a few seconds the clumping of particles observed in the drop indicates the presence of virus. This test has been successfully applied in India with dolichos enation mosaic virus (Badami, 1959). In the field chirke mosaic of cardamom and cassava mosaic have been screened using respective antisera (Ganguly *et al.*, 1970).

Detection of latent, unknown new viruses: DeBruyn Ouboter (1952) used a potato variety 'Light Industries' as the source of inoculum of potato virus A to prepare an antiserum, but instead she obtained an antiserum which reacted positively with sap from plants of several varieties including plants which were known to be free from potato virus A. Most of the potato plants were free from obvious symptoms. This led to the discovery of a new latent virus named as virus 'S' in honour of Professor Slogteren.

Recognition of relationship between viruses: The basis for serological relationship is sharing of common antigen, hence an antiserum prepared against one virus will react with the sap of any plant infected with that virus (homologus) or its strain, provided the virus content is adequate, but not with the sap of any plant infected with a different virus.

Bawden and Pirie (1937) showed that cucumber virus 3 and 4 had few antigens in common to TMV. These serologically differed considerably from other strains of TMV. Badami (1959) reported that TMV and dolichos enation mosaic virus (DEMV) are serologically unrelated. He also reported in 1963 that Ellen Ball from Nebraska (USA) using the complement fixation and agar gel diffusion tests, found that the southern bean mosaic virus (= bean virus 4 and 4A) and DEMV are serologically unrelated but at Rothamsted Experiment Station, positive serological relationship

between DEMV and bean form of Nigerian cowpea virus was established.

Bawden (1958) has shown that the Nigeria cowpea virus (Lister and Thresh, 1955) and the southern sunnhemp mosaic (Capoor, 1950) are the strains of TMV. Bartels (1958) attempted serological relationship among strains of potato virus Y and showed that they are neither identical nor closely related.

Serological tests have been made by using tube precipitin, slide agglutination, or double gel diffusion methods. Most of the workers in India (Anand *et al.*, 1961; Chenulu *et al.*, 1968; Ganguly *et al.*, 1970; Shankar *et al.*, 1969) have reported only homologous reaction.

A few workers have also shown the serological relationships of different viruses, namely, southern sunnhemp mosaic, bottlegourd mosaic (BGMV), cowpea (Chavali) mosaic and tobacco mosaic viruses (Anand and Sahambi, 1965; Nariani *et al.*, 1970); TMV from potato and common strain of TMV (Phatak and Verma, 1967); BGMV and watermelon mosaic virus (Shankar *et al.*, 1969); petunia mosaic and tobacco ringspot viruses (Rani *et al.*, 1969); cowpea chlorotic spot, southern sunnhemp mosaic, tobacco mosaic and dahlia mosaic viruses (Sharma and Verma, 1975). The following viruses have been observed to be serologically different, namely, Henbane mosaic virus and TMV (Chenulu *et al.*, 1968); Tori mosaic virus and BGMV (Mitra and Nariani, 1965); Snake-gourd mosaic virus and BGMV (Shankar *et al.*, 1969). Barley stripe mosaic and barley mosaic viruses (Dhanraj and Raychaudhuri, 1969). Ganguly *et al.* (1970) have obtained successfully the antiserum of cassava mosaic virus which is transmitted by white flies.

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6

Detection at Seed and Cellular Level

There are various techniques employed for detecting the presence of virus infections, namely,

- (a) Symptomatology.
- (b) Local lesion host/indicator plant,
- (c) Seed morphology,
- (d) Serology,
- (e) Electron microscopy,
- (f) X-ray of true seeds,
- (g) Staining techniques,
- (h) Fluorescent microscopy,
- (i) Fluorescent antibody technique and
- (j) Fluorescent staining for nucleic acid.

However, not a single method should be accounted for conclusion and a combination of some of the above methods is essential for detection.

The routine methods namely, symptomatology, indicator hosts, serology, electron microscopy are time consuming compared to seed morphology or staining techniques used for detecting the presence of virus at seed level.

McLintock (1917) presented the first evidence that plant viruses are transmitted through seed in the case of cucumber mosaic and lima bean mosaic viruses. The evidence was more suggestive than conclusive. Reddick and Stewart (1919) and Doolittle and Gilbert (1919) confirmed the earlier findings. Seed transmission of a number of other viruses was demonstrated in the next few years. So far fifty or more viruses are known to be seed transmitted. The problem of why there are not many is still not entirely solved. It is not well understood why at times only a specific host permits passage of the virus through its seed

while others do not, e.g., tobacco mosaic virus in seeds of tomato and pepper. Upto 100 per cent transmission was reported in seed of individual soybean plants infected with tobacco ringspot virus (Athow and Bancroft, 1959). Lettuce infected with lettuce mosaic may produce 3 to 10 per cent of the seed carrying the virus (Newhall, 1923; Couch, 1955). Variation in rate of seed transmission may also be due to the stage at which the plants become infected.

SOME OF THE SEED TRANSMITTED VIRUS DISEASES

<i>Name of virus</i>	<i>Host</i>	
Alfalfa mosaic	<i>Capsicum annuum</i>	Sutic (1959)
Barley stripe mosaic	<i>Hordeum vulgare</i>	McKinney (1951)
	<i>Triticum aestivum</i>	McNeel and Afanasiev (1955)
Bean mosaic	<i>Phaseolus vulgaris</i>	Reddick and Stewart (1919)
Cowpea mosaic	<i>Vigna sinensis</i>	McClean (1941)
Tobacco mosaic virus	<i>Lycopersicon esculentum</i>	Bewley and Corbett (1930)
	<i>Capsicum frutescens</i>	McKinney (1952)
Tobacco ringspot	<i>Nicotiana tabacum</i>	Valleau (1932)
TRSV	<i>Petunia hybrida</i>	Henderson (1931)
	<i>Glycine max</i>	Desjardins <i>et al.</i> (1954)
Southern bean mosaic	<i>Vigna sinensis</i>	Shephard and Fulton (1962)
Cherry ring spot	<i>Prunus avium</i> var. mazzard	Cochran (1946)

ELIMINATION OF VIRUS FROM SEED

With most seed transmitted viruses there is good evidence that the virus is carried internally. Heat treatment of infected seed does not eliminate the virus. Well-dried seed is surprisingly resistant

to high temperature. Virus in such seeds can tolerate as much heat as the seed tolerates suggesting that the virus is less hydrated than in the sap and thus acquires greater resistance to heat denaturation.

Chamberlain and Fry (1950) and Taylor *et al.* (1961) found that most of the virus (TMV) present with tomato seed is in the seed coat. The external infection can be eliminated by acid extraction or trisodium phosphate treatment.

Mechanism restricting seed transmission

Two mechanisms may operate to prevent seed transmission (i) elimination of virus from embryo of mature seed and (ii) exclusion of virus from developing seed (there must be some barrier preventing virus movement between developing seed and the mother plant).

DETECTION PROCEDURES

Seed Morphology

It is observed that at times the virus affected seeds differ morphologically from the healthy ones.

Small and deformed seeds often give rise to weak seedlings. In some cases seeds from virus affected crops are small in size and also deformed. It has been observed in the case of barley false stripe virus in barley seeds and cowpea mosaic in cowpea seeds that a correlation could be established between seed morphology and seed transmission of barley false stripe virus and cowpea mosaic virus in barley and cowpea respectively (Phatak and Summanwar, 1967). Similar observations have been reported by Kennedy and Cooper (1967) concerning the association of virus infection with mottling of soybean against soybean mosaic virus.

As such seed morphology serves as one of the criteria for detecting the presence of virus at seed level.

Seed Germination: Viruses can be diagnosed within a reasonable limit by growing seedlings at high temperatures and bright light (Afanasiev, 1956; Hampton *et al.*, 1957). The tests have been successfully carried out in the United States in Kansas and Montana. Phatak (1974) observed typical symptoms of barley stripe mosaic virus (BSMV) in one week old seedlings. He also developed a 'blotter test' in petridishes for four NEPO viruses in *Petunia violacea*.

Testing on Indicator plants: Pelet and Gagnelin (1963) and Pelet (1965) made use of *Chenopodium guinea* for indexing seed-borne lettuce mosaic virus. Marron and Messian (1967) improved the test for seed health testing. Phatak (1974) found that by using this test a single infected seed in a batch of 300, uniformly results in infection of *C. guinea*.

Electron Microscopy: Gold *et al.* (1954) found with an electron microscope particles of BSMV in embryo and endosperm of individual seeds of barley and wheat. They reported that the testing is practical and convenient. Walkey and Webb (1970) reported that the particles of cherry leaf roll virus could be seen in tubular inclusion bodies in homogenates of mature seeds of *Nicotiana rustica*. Phatak (1974) found particles of barley chlorotic spot virus (BCMV), BSMV and soybean mosaic virus (SMV) in phunule extracts of barley, bean and soybean respectively. However, he observed that electron microscopy is tedious for routine testing.

Serology: Some serological results have been used for diagnosis with encouraging results. Scott (1961); Hamilton (1965); Phatak (1974) have reported promising results with Ouchterlony test for detecting tobacco mosaic virus (TMV) contaminating tomato seed. Small batches of seed (0.1-1g) were ground in 0.5 to 5 ml of normal saline (0.85 per cent) and resultant extracts were distributed in wells facing the central antiserum depot. Characteristic bands were visible between wells charged with TMV containing extracts and the antiserum depot, within less than twenty-four hours. This test is used in Chile for detecting squash mosaic virus (SMV) in imported cucurbit seeds.

DETECTION OF VIRUS DISEASES AT CELLULAR LEVEL

Fluorescent Antibody Technique

This technique developed by Coons *et al.* (1942) has been employed in the study of a number of viruses (Coons, 1958; Schramm and Rottger, 1959; Nagajay and Black, 1961). At IARI, it has been successfully used in detection of mycoplasma in greening affected citrus plants (Kumar *et al.*, 1974). The antiserum is obtained in a routine manner and gamma-globulins extracted from the antiserum is conjugated with fluorescein isothiocyanate (FITC). For staining sections of infected as well as uninfected leaves are flooded with tris-HCl buffer before covering each with 1-2 drops of FITC

conjugated antiserum on slides and leaving them in a moist chamber for eight to twelve hours at room temperature. The stained sections when viewed under fluorescent microscope, healthy leaf sections give dull blue auto-fluorescence while the diseased leaf sections give brilliant apple green fluorescence. Phatak (1974) has attempted fluorescent antibody test for soybean mosaic virus in soybean plumules. He reported encouraging results but added that because of its high sophistication, the test does not qualify for routine testing yet.

Fluorescent Staining for Nucleic Acid

Efforts are being made in various laboratories to develop techniques of quick detection of plant viruses. In that connection large number of stains have been employed by various workers for differentiating nucleic acids in plant viruses and thereby detecting the virus infection. Stains such as Giemsa (Bald, 1949), phloxin (Rubio-Huertos, 1962), trypan blue (McWhorter, 1941), acridine orange (Hirai and Wildman, 1963, Hooker and Summanwar, 1964), 2, 3, 5-triphenyl tetrazelium chloride (Antoine, 1958), etc., have been employed for detecting virus infections in various vegetative plant tissues. Besides these, some other simple chemicals have been used for giving specific colour reactions. No standard method however, is applicable in a general way. It has been found in our laboratory that Phloxin-trypan blue, methyl green-pyronin and acridine orange stains with the help of fluorescence microscope are very useful in finding out differences between healthy and virus infected cells. Raychaudhuri and Verma (1966) have tested methyl green-pyronin stain combination on TMV infected and healthy tobacco callus cells. RNA content was found to be increased in TMV affected cultured cells. This may be due to the presence of viral RNA, which is stained red in the cytoplasm. Similarly when epidermal peelings of cowpea leaves infected with CpMV were stained large irregularly shaped cytoplasmic inclusions were observed. These inclusions were very prominent and completely surrounded the nucleus (Summanwar *et al.*, in press). When Phloxin-trypan blue combination was used against the same host cowpea inclusion bodies were stained well and appeared deep purple. Epidermal peelings obtained from diseased leaves of large cardamom (*Amomum subulatum* Rob.) when mixed with phloxin and trypan blue showed the

presence of inclusions adjacent to the nucleus, whereas no such inclusions were observed when epidermal peelings from healthy leaves were stained (Phatak and Summanwar, 1967). Kripa Shankar while working at IARI observed that yellow mosaic virus disease of mung brings about a distinct change at cellular level in infected *mung* leaves (*Phaseolus aureus* Rob.) using pyronin C-methyl green stain combination. It has been shown that diseased nucleus gets enlarged and is surrounded by red stained amorphous cytoplasmic inclusion bodies. At the same time cell walls also get thickened compared to healthy cells.

Acridine orange (AO.), a fluorescent dye when used as a stain with UV light as source for illumination gives better and clear picture in the cytopathological studies. Because of its differential affinity for nucleic acids AO. differentiated RNA and DNA of the cell and could be used for the study of virus infected plant cells.

Hirai and Wildman (1963) reported the use of AO in their cytopathological studies of tomato hair cells infected with tobacco mosaic virus. RNA was characterised by the presence of brick red fluorescence and yellow fluorescence indicated the presence of DNA. Similarly using AO and a fluorescent microscope a large number of host and virus diseases were studied at IARI, Mycology Division, to study its usefulness for the detection of viruses at cellular level. Cowpea mosaic virus in cowpea seed was studied in detail. In epidermal peelings infected cells were full of bright red fluorescing material surrounding the nucleus indicating concentration of virus material. When seeds from infected plants were allowed to germinate and the sections of their radicals (T-5) were treated with AO and viewed in UV microscope, it revealed that wherever dense red fluorescence was observed those seeds were carrying the virus. This was confirmed by inoculating the remaining portion of the seed on indicator plants. This technique could be used for detecting the seed borne viruses to limited extent, because the technique did not differentiate host nucleic acid and viral nucleic acid.

This technique was also useful in large cardamom diseases like chirke and foorkey and sugarcane grassy shoot where there was distinct difference between healthy and diseased cells.

Recent advances in freeze-etching and freeze-drying of specimens in preparation for electron microscopy make it possible to

clearly visualise surface subunit and internal structure of virus particles for their detection. Stereo-electron microscopy aids in the interpretation of such specimens. Electron micrographs reveal the structure of virus particles, both *in vivo* and in purified preparation and reveal some of the developmental stages of virus particles within the infected cells (Stoorc, 1974).

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7

Tissue Culture

Tissue culture has been used extensively in the study of virology. Culture collection and maintenance of plant viruses is possible by isolating calli of plant species which are already affected with viruses showing typical symptoms. A number of chemicals have been screened effectively under controlled conditions for their anti-viral activity *in vitro*. The most important object of plant virologist is to obtain virus free seeds and seed stocks particularly in case of host species which are vegetatively propagated. This can be achieved by callus, embryo, meristem, budwood and nucellar cultures.

MAINTENANCE OF PLANT VIRUS CULTURES

In places of extreme climatic conditions it is not possible to maintain plant viruses in glasshouses round the year. Plant tissue culture is found to be useful in maintaining some virus cultures under controlled conditions (Mishra and Raychaudhuri, 1967). The stem, petiole and leaves of virus infected plants are used as a source for obtaining calli. These cultures are maintained through regular transfers of calli to fresh media.

Four different viruses, namely, chilli mosaic virus, sunnhemp mosaic virus, potato virus X and tobacco mosaic virus were maintained for two years by serial transfers without any loss of infectivity on White's simple agar medium (Raychaudhuri and Mishra, 1965b; Mishra *et al.*, 1964). Pumpkin mosaic virus which has a longevity *in vitro* of eight hours only and host range restricted to cucurbitaceae could be maintained for six months.

The differentiated shoot cultures have more virus concentration than undifferentiated calli (Mishra and Raychaudhuri, 1968).

The initial infectivity of the callus cultures depend on their source and thus the callus obtained from leaf parenchyma has maximum virus concentration as compared to the tissues from stem (Raychaudhuri and Mishra, 1965b). However, when the calli obtained from stem, petiole and leaf of cowpea plant infected with cowpea mosaic virus were assayed, no significant difference in the viral concentration was observed in the inocula obtained from the calli of the above series.

Nutritional and Therapeutic Studies

Viruses are nucleoproteins and obligate parasites. They do not possess any independent metabolic system. Therefore, almost any change in nutritional as well as environmental conditions, affect their infectivity. The tissue culture technique thus provides an interesting tool for investigations on the nutritional and other environmental requirements in the virus infected host tissue (Raychaudhuri, 1966).

Anti-Viral Chemicals

Various groups of chemicals, namely, growth regulators, antibiotics, metabolites, anti-metabolites, aminoacids, aflatoxins, tannins and surfactants have been screened by incorporating in media on which the virus infected tissue cultures were grown.

Many growth regulators such as indolebutyric acid, indoleacetic acid, gibberellic acid and 2, 4-dichlorophenoxyacetic acid were found to reduce the concentrations of tobacco mosaic virus, cowpea mosaic virus, potato virus X and potato virus Y in tissue culture (Raychaudhuri, 1966; Mishra *et al.*, 1964).

The effect of some metabolites and their analogs on the infectivity of tobacco tissue culture containing chilli mosaic virus was also studied by Raychaudhuri and Mishra (1965a) besides various other chemicals such as methionine, arginine, ethionine and lysine. Results suggested that the incorporation of purine and pyrimidine bases in the medium enhanced the infectivity whereas the corresponding analogs as thiouracil, benzimidazol, etc., retarded its infectivity.

Earlier Kassanis (1957a) found that the incorporation of uracil alone into the medium doubled the virus concentration. The addition of sodium salt of RNA, however, decreased the virus concentration but increased the growth of the tissue when

incorporated at 0.1 g/litre level. A similar increase in growth occurred with DNA as well as adenine. Kassanis (1957a) further studied the effect of incorporating TMV in the autoclaved and unautoclaved media. TMV added before autoclaving decreased growth of the tissue, but increased the virus concentration. Kurtzman (1959) and Kurtzman *et al.* (1960) working on the inhibition and multiplication of TMV in tobacco tissue cultures, used analogs of the nucleic acid bases, particularly purines. Of these compounds, caffeine at low concentration stimulated the synthesis of TMV but decreased the growth of the tissue.

Kassanis (1957a) studied the effect of phosphate concentration on TMV infectivity in a modified medium. Increasing phosphate content in the medium increased the growth of the tissue.

The inhibitory effects of anti-viral chemotherapeutants were also tested by incorporating them in tissue culture medium. Blastocidin, aflatoxin (Subbarayudu *et al.*, 1969), surfactants, tannic acid, ellagic acid (Verma and Raychaudhuri, 1968) and plant extracts (Gupta and Raychaudhuri, 1971 and 1972a) were found to reduce the virus concentrations of TMV, CpMV, PVX and PVY. In tissue culture studies, gallic acid and gallotannin showed more inhibition of virus multiplication. Ellagic acid and catechin showed less inhibition of PVY (Gupta and Raychaudhuri, 1972b).

Radiation Studies

Radiation therapy of plant viruses in tissue culture offers the possibility of utilising this method for obtaining virus-free stocks. Incorporation of chemicals in the medium followed by gamma irradiation of the tissues has been found to give additive effect by inactivating more virus particles than by either treatment singly. Such combined effects were noticed with growth regulators (GA and 2, 4-D) and irradiation in the case of TMV. PVY infectivity was reduced when the infected tissues were cultured on the medium irradiated by gamma rays. Rao while working at IARI obtained complete inactivation of CpMV when infected calli were grown on medium containing sodium lauryl sulphate (100 ppm) and irradiated with gamma rays.

Studies conducted to see the effect of radioactive phosphorus and sulphur on a strain of TMV (CPO strain) in tissue culture showed that there was greater uptake of isotopes by virus affected tissues as compared to healthy ones, when grown on a medium containing

the radio-isotope at an activity of 15 microcuries per ml. Quantitative data on uptake showed that it was more in differentiated tissues than in callus only. Radioactive phosphorus at this dose had no effect on the infectivity, while radioactive sulphur at the same dose inhibited the infectivity by about 90 per cent (Chatrath *et al.*, 1970).

VIRUS FREE CULTURES

Apple mosaic virus and citrus exocortis are not known to be transmitted by insect vectors. Virus free plants from such sources obtained under controlled conditions employing tissue culture are of great use in raising new orchards. Buds from mosaic affected apple plants, budwood from citrus plants affected with exocortis, tristeza and greening diseases were grown on synthetic media. A basal medium (Murashige and Skoog, 1962) was used for growing virus free citrus buds from the tristeza and greening affected budwood and developed into plantlets. However, the transplanted plantlets having short roots did not survive.

Embryo Culture

Embryo culture studies of virus affected runner bean, were conducted and the presence of virus was detected by bioassay on indicator plants and from the symptoms shown in the differentiated plantlets (Mishra *et al.*, 1967). Cowpea embryos obtained from seeds of CpMV infected cowpea plants were grown on Murashige and Skoog's basal medium supplemented with sodium lauryl sulphate. Only in few embryos the virus could be detected.

Raychaudhuri and Verma (1966) stained healthy and TMV infected tobacco calli with pyronin-methyl green. The TMV infected cells were deeply stained showing that RNA increased in the affected tissue which seems to be due to the presence of viral RNA.

Outstanding work has been done on ovule, ovary and nucellar cultures (Maheshwari, 1958; Rangaswami, 1961; Maheshwari and Baldev, 1961). Some of these results are important for plant virologists especially for studying the nature of seed transmission of viruses. Crowley (1959) proved that absence of seed transmission of some of the highly infectious plant viruses is due to their inability to infect and survive in the young gametophytic and meristematic tissues. The culture of nucellar embryos can be used as a means of freeing citrus clones of viruses (Weathers and Calavan, 1959).

With the exception of xyloporosis in sweetlime, most viruses found in citrus are not transmitted through seeds. Nucellar tissue in most of these cases is virus-free and can be grown into fully matured plants by freeing them from their integuments and growing them on an appropriate nutritive medium.

Meristem Cultures

Many viruses fail to invade the growing tips of new shoots. This may be either due to slow movement of the virus or to high concentration of auxin.

Holmes (1948) freed a number of dahlia varieties from tomato spotted wilt virus by grafting the meristematic tips on healthy stocks and subsequently rooting them in soil. Sometimes the virus invades the tip with the exception of actual meristem. In such a case only meristem is taken out and grown on culture medium. Morel and Martin (1952, 1955) produced healthy stocks of some virus affected varieties of dahlia and potatoes by excising and growing the meristem in a sterile synthetic medium. These cuttings developed into small plantlets with well developed root systems. When they become large enough, they were transferred to soil and virus free plants were thus obtained (Kassanis, 1957b).

Some of the workers obtained virus free stocks by heat treatment before culturing the meristems. Quak (1957) obtained virus free stocks by isolating some of the leaf-primordia from plants held for 6 to 8 weeks at 40°C and culturing them on nutrient media. In India virus free cane was obtained by the culture of stem apices in nutrient media (Mascarenhas *et al.*, 1973). Shoot apices (0.5 to 1.5 mm long) excised from virus infected sugarcane plants var. Co 740 were grown aseptically in liquid media containing mineral salts, vitamins and glycine, sucrose, gibberellic acid, coconut milk and indole butyric acid. When the shoot apices grew to about 2 cm, they were transferred to a White's liquid medium in which they developed roots.⁹ These plants were then transferred to soil and grown to maturity. Over 2000 virus free plants were raised by this method.

Single Cell Culture

The idea of culturing single cells in isolation was set forth originally by Haberlandt in 1902. Muir *et al.*, (1954, 1958) placed single

cells on pieces of filter paper, which in turn were transferred to the top of established nurse cultures. Torrey (1957) placed single cells in a ring around a large nurse culture. Jones *et al.*, (1960) have developed single cell cultures in a drop of liquid medium submerged in mineral oil.

By using the microchamber technique, healthy and TMV infected tobacco cells were observed under phase contrast microscope. The TMV (CPO strain) infected tobacco cells had three types of inclusion bodies, namely, round, needles and hexagonal crystals, apart from normal cellular constituents.

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Virus Inhibitors

Some earlier investigations on plant viruses indicated clearly the existence of inhibitors in plant juices that irreversibly inactivate plant viruses. Also various other agents such as growth products of bacteria and fungi, pyrimidine and purine analogues, inorganic salts, proteins, enzymes, polysaccharides, sera, aminoacids and insect juices are known to inhibit plant viruses. Gupta (1957) has discussed some important features of the inhibition and alteration of viral infectivity in plants. Raychaudhuri (1960, 1961) and Verma *et al.* (1974) have reviewed the work on plant virus inhibitors with special references to some viruses occurring in India.

PLANT EXTRACTS

Plant virus inhibitors have been reported to be present in juices of plants like pokeweed (Dugger and Armstrong, 1925), chilli (Hirai, 1849; Vasudeva and Nariani, 1952; Sharma and Raychaudhuri, 1956; Prasad and Raychaudhuri, 1961; Paliwal and Nariani, 1965), coconut milk and copra extract (Gendron, 1950), datura, potato and black nightshade (Vasudeva and Nariani, 1952; Paliwal and Nariani, 1965), cucumber and watermelon (Sill and Walker, 1952).

Chilli extract is very effective in inhibiting potato virus X, radish mosaic, bottlegourd mosaic and zinnia mosaic viruses while datura extract effectively inhibits potato virus X and bottlegourd mosaic viruses. Paliwal and Nariani (1965) reported that leaf extracts of *Capsicum annuum*, *Datura stramonium*, *Beta vulgaris*, *Chenopodium album*, *Sensbiera didyma*, *Gynandropsis pentaphylla*, *Dianthus caryophyllus*, *Prosopis juliflora* and latex from *Carica*

papaya were strong inhibitors of sunnhemp mosaic virus. The inhibitors were found to be present in roots, leaves and fruits of *C. annuum* and roots and leaves of *B. vulgaris* and *Raphanus sativus*. Leaf extract of *Acacia arabica* was found to inhibit PVY infectivity *in vitro* (Gupta and Raychaudhuri, 1971a). Leaf extracts of *Callistomon lanceolatus*, *Syzygium cumini* and *Acacia arabica* showed effective inhibition of PVY. Stem, bark and leaf extract of *Cinchona ledgeriana* and pericarp extract of the fruits of *Terminala chebula* and *T. belerica* gave comparatively less inhibition (Gupta and Raychaudhuri, 1971b, 1972b). Leaf extracts of *Callistomon lanceolatus* and *Syzygium cumini* inhibited the local lesion production when mixed with potato virus X infective sap (Gupta and Raychaudhuri, 1972c). Four cinchona alkaloids, namely, cinchonine, cinchonidine sulphate, quinine sulphate and quinidine sulphate function as chief antiviral principle for potato virus X (Verma and Raychaudhuri, 1970a). The dry fruit pericarp of *T. chebula* has been reported to contain tannins, namely, chebulinic acid, tannic acid and gallic acid which are strong inhibitors of potato virus X (Verma *et al.*, 1969; Verma and Raychaudhuri, 1970b).¹

Microbial growth products are found to be very effective in inhibiting the viruses. Johnson and Hoggen (1937) were the first to investigate the inhibition of plant viruses by fungal growth products. Johnson (1938), Fulton (1943), Ramon *et al.* (1948), Gupta and Price (1950), Gendron (1950), Gupta and Price (1952), Bawden and Freeman (1952), Slagle *et al.* (1952) and Sharma and Raychaudhuri (1956) have also reported active inhibitors in microbial growth products.

Growth products have been obtained from *Aspergillus niger*, *Trichothecium roseum*, *Fusarium avenaceum*, *Colletotrichum lindemuthianum*, *Rhizoctonia solani*, *Helminthosporium halodes* and *Bacillus subtilis*. Appreciable inhibition was noticed in case of growth products of *Aspergillus niger* on radish mosaic virus, *Trichothecium roseum* on potato virus X and radish mosaic virus and *Bacillus subtilis* on radish mosaic and vinca mosaic viruses. Kang, Raj and Chohan (1972), reported seventy to eighty per cent inhibition of vegetable marrow mosaic virus by the filtrate of aflatoxin-producing *Aspergillus flavus* isolates.

SYNTHETIC CHEMICALS

Chemotherapeutic treatments for inhibition of plant viruses have been studied with success. Thiouracil has been shown to suppress the rate of multiplication of tobacco mosaic virus (Commoner and Mercer, 1951, 1952; Nichols, 1953, 1954; Bawden and Kassanis, 1954; Holmes 1955), a ring spot strain of potato virus X (Sharma and Raychaudhuri, 1956; Raychaudhuri and Sharma, 1962), and the virus causing 'chirke' disease of large cardamom (Raychaudhuri and Chatterjee, 1961). Four compounds, namely, gallic acid, gallotannins and ellagic acid were tested for their inhibitory effect on PVY *in vitro* and in tissue culture. First two showed inhibition of PVY infectivity *in vitro*. The other two showed less inhibition (Gupta and Raychaudhuri, 1972a).

Raychaudhuri and Mishra (1963) reported that out of the six growth regulators, namely, α -naphthyl acetic acid, indole acetic acid, indole butyric acid, phenyl acetic acid, gibberellic acid and 2-4-dichlorophenoxy acetic acid tested *in vitro*, α -naphthyl acetic acid was the most effective inhibitor of sunn hemp mosaic virus (SMV). The efficiency of gibberellic acid in reducing the infectivity of SMV is enhanced appreciably when it is mixed with equal proportion of glutamic acid both being employed at a concentration of 0.001 per cent. Gibberellic acid reduces the infectivity *in vitro* of chilli mosaic virus (ChMV) to the extent of 46 per cent when used at a concentration of 0.001 per cent, in solution. Other growth regulators, namely, 2, 4-dichlorophenoxy acetic acid, α -naphthyl acetic acid, indole butyric acid, indole acetic acid and phenyl acetic acid are less efficacious (Raychaudhuri and Mishra, 1964).

Coumarin inhibited the infectivity of tobacco mosaic virus. Pre-inoculation root-dip of tomato seedlings, a systemic host of virus, delayed the expression of symptoms. Pre-inoculation and post-inoculation spraying of *C. amaranticolor* leaves was equally effective in inhibiting the local lesion formation. Further, reduction of the local lesion formation by about ninety-two per cent was found when *Datura stramonium* plants were root dipped for six hours, in coumarin at 25 ppm before inoculation (Mishra *et al.*, 1968).

A number of growth regulators and related substances are known to have inhibitory effect on the development and concentration of plant viruses *in vivo* and *in vitro*. Bhatt and Verma (1958) reported recovery of tomato streak by spraying naphthalene

acetic acid and indole butyric acid. It was noticed that methyl acetic acid was most effective and inhibited the sunnhemp mosaic virus upto 78.51 per cent (Raychaudhuri and Mishra, 1963). Few more growth regulators such as phenyl acetic acid, indole butyric acid and indole acetic acid were also found to have inhibitory effect on sunnhemp mosaic virus.

Badami (1959) obtained insignificant inhibition of a SMV strain as compared to strains of TMV. Upreti *et al.* (1954) observed that PVX and PVY are significantly inhibited when thiouracil is mixed with inoculum. Mukherjee and Raychaudhuri (1966) observed that if leaf curl affected tomato scions were treated with thiouracil solutions and then grafted on test stocks, resulted in significant disease reduction presumably due to inactivation of the virus. Thirumalachar *et al.* (1973) reported reduction in disease incidence and uptake of virus by white flies using 75 ppm DPB as foliar sprays given at 15 days intervals. It also increased yield of tomatoes by 50 per cent.

Raychaudhuri and Mishra (1964, 1968) observed that indole acetic acid at 0.001 per cent could inhibit *in vitro* multiplication sunnhemp mosaic virus and chilli mosaic virus upto 40 and 30 per cent, respectively. Upreti *et al.* (1964) reported upto 58 and 47 per cent inhibition *in vitro* of PVX and PVY, respectively, by employing IAA at 50 and 15ppm. Mitra while working with the author at the IARI obtained inhibition of TMV and PVY *in vitro* and *in vivo* in different degrees by various modes of NAA application. Chowfla in 1972 observed inhibition of broad bean mosaic virus as also delay in symptom expression with NAA.

Kinetin and nitroureacil have been found to be very good inhibitors of cowpea mosaic virus *in vitro* by Phatak in 1968.

Subbarayudu and Raychaudhuri (1969) tested nine growth regulators for their effect on the infectivity of the tobacco mosaic virus. Although none of them indicated complete inhibition of the virus. IBA, 2, 4-D, GA, IAA and PAA were found to be significantly effective in reducing the viral infection. Different concentrations of IAA, NAA and DL-2 amino butyric acid showed significant differences in reducing the viral infection. Colchicine indicated increased infectivity at concentrations of 1, 10 and 100 ppm except at 1000 ppm.

Cytovirin has shown to be most effective antibiotic against cowpea mosaic virus *in vitro*. Inhibition of infectivity of SMV

and PVX in local lesion hosts has also been observed at the IARI.

Joshi in 1970 studied twelve chemicals belonging to four groups, namely, dyes, base analogues, antibiotics and growth regulators, *in vitro* and *in viro* for their effect on the infectivity of PVY. All the chemicals showed antiviral effect to varying degrees. Among the dyes, acridine orange and crystal violet were superior. Base analogues, e.g., 5-bromouracil, 5-nitouracil; antibiotics, e.g., blasticidine, chloromycetin, terramycin, and growth regulators, e.g., phenyl propionic acid and 2, 4-D showed inhibitory effect on PVY. Hariharasubramanian (1968) observed that daily treatment with 2, 4-D and two base analogues starting from twenty-four hours after inoculation, inhibited dolichos mosaic virus in *Dolichos lablab*.²

Loring (1942) demonstrated a strong inhibitory effect of pancreatic ribonuclease (RNase) on the infectivity of TMV. Badami (1959) observed that when equal volumes of 50 ml/litre solution of pancreatic ribonuclease were added to sap from plants infected with either spinach or yellow strain of CMV, the mixtures were made almost non-infective towards tobacco and french bean. It was found that the infectivity was regained when the mixtures were suitably diluted with water indicating thereby that the decrease in infectivity caused by the enzyme did not result from hydrolysis of the virus nucleic acid.

Nene and Thornberry (1969, 1970a, b) reported the inhibitory effect of carboxymethylated ribonuclease on tobacco mosaic virus and found that the inhibition was host dependent indicating that the enzyme acted on the host rather than the virus. It was suggested that the enzyme adsorbed to the infection sites excluding virus attachment.

Chowfla at the IARI in 1972 tested pancreatic ribonuclease and venom phosphodiesterase enzymes for their effects on the infectivity of broad bean mosaic virus (BBMV) and obtained 91.65 and 54.95 per cent inhibition of the virus respectively when the enzymes were mixed with the virus inoculum at 1,000 ppm.

Nagpal in 1973 tested these enzymes on bottlegourd mosaic virus (BGMV). RNase was found to be a very effective inhibitor of the virus, not only *in vitro* but also affected the systemic development of the disease. The inhibition was not affected by changes in virus dilution but decreased significantly with the time of storage of virus-enzyme mixture. When sprayed on bottlegourd plants

at the concentration of 1,000 ppm for three consecutive days before inoculation, it caused delay of one week in the symptom appearance and a reduction of 51.64 per cent in the virus content of the plants. When the enzyme was applied after inoculation the effect seemed to be considerably less.³

IONISING RADIATION

Nariani and Paliwal (1963) reported complete inhibition of sunn-hemp mosaic virus in sap by gamma radiation at the dose of 300,000 roentgens. Subbarayudu and Raychaudhuri (1969) reported an additive effect in reducing TMV concentration in tobacco calli grown in a medium containing GA and 2, 4-D, when exposed to gamma radiation. Mitra and Raychaudhuri (1971) observed a reduction in viral infectivity of PVY when the callus was grown on irradiated medium or when the irradiated callus was grown on fresh medium. Phatak in 1968 reported from the IARI reduction in the multiplication of CpMV when the infected callus culture was irradiated with gamma rays and X-rays at 100 kR and 30 kR, respectively.

Ultraviolet irradiation has been proved to inhibit plant viruses and was first demonstrated by Finmen and Breyer (1963). Since then several investigators have shown inhibitory effect of ultraviolet irradiation (Mulvania, 1926; Arthur and Newell, 1929; Stanley, 1934; Price and Gowen, 1937; Bawden and Pirie, 1938; Lee and Smith, 1940).

In India it has been observed that the chilli mosaic, bottlegourd mosaic, zinnia mosaic, periwinkle mosaic and soybean mosaic viruses are completely inactivated when virus extracts are exposed at 514.00 m.u.d., 160.00 m.u.d., 160.00 m.u.d. 80.00 m.u.d. and 160.00 m.u.d. respectively to ultraviolet irradiator (Jha and Raychaudhuri, 1956; Raychaudhuri *et al.*, 1950; Prasad and Raychaudhuri, 1962; Joshi and Raychaudhuri, 1964; Nariani and Pingaley, 1960). However, cowpea mosaic virus was not completely inactivated even after exposure to 240 m.u.d. (Nariani and Kandaswami, 1961). Irradiation of leaves of local lesion hosts by ultraviolet rays (20.00 m.u.d.) immediately after they were inoculated prevented lesion formation to the extent of 96.5 and 86.5 per cent in the case of radish mosaic virus and PVX respectively (Sharma and Raychaudhuri, 1956; Raychaudhuri and Prasad, 1965).

Combined effects of chemotherapy and gamma radiation on tobacco mosaic virus and cowpea mosaic virus have been studied in tissue culture (Subbarayudu and Raychaudhuri, 1969). Callus cultures containing virus, when treated individually with GA, 2, 4-D, SLS and aflatoxin and then exposed to gamma radiation showed a further decrease in viral concentration than was noticed either by chemical or by radiation treatment alone.

When P_{32} and S_{35} were separately added to the medium on which the callus cultures were grown, there was a greater uptake of isotopes by TMV affected tissues as compared to healthy ones. Quantitative data on uptake showed that it was more in differentiated tissues than in callus only. Further the virus concentration was reduced by 90 per cent by S_{35} but not by P_{32} .

SURFACTANTS

On the basis of many qualitative observations it has been indicated that synthetic detergents produce diverse effects on proteins and biological systems (Putman, 1948). Ansen (1939) observed that a variety of detergents denature haemoglobin at the isoelectric point and keep the denatured proteins in solution. Bull and Neurath (1937) first noted the precipitation of egg albumin by sodium dodecyl sulphate (SDS). Vaccinia virus is inactivated by SDS and few other detergents (Klein *et al.*, 1945).

The hydrogen sulphate of dodecyl alcohol inactivated potato virus X and tomato bushy stunt virus and, it separated nucleic acid from protein (Bawden and Pirie, 1938). Effect of SDS was also studied by Sreenivasaya and Pirie (1938).

Brakke (1959) screened number of detergents for dispersing the aggregated barley stripe mosaic virus (BSMV). Detergent Igepon T-73 was found to be the best by estimating the infectivity of the virus than other detergents used.

Lifdner *et al.*, (1959) found that cationic types were more effective than anionic or non-ionic surfactants tested against TMV infection. Schneider and Mitchel (1962) reported that dodecyl sodium sulfosuccinate inhibited TMV and SBMV.

Phatak while working at IARI in 1968 obtained 100 per cent inhibition of cowpea mosaic virus (spherical) with Teepol B-300, Tween-20 and Tween-80 and, the chemicals remained effective upto twelve hours after virus inoculation.

Rao and Raychaudhuri (1973) while screening twelve surfactants, found sodium lauryl sulphate (SLS), Idet-20, emulin and daichi to be highly effective in inhibiting cowpea mosaic virus (CPMV) infection. SLS at 1000 ppm when mixed with the inoculum *in vitro* completely inactivated the CPMV and CMV.

Nagpal observed that Idet-20 and SLS cause a high degree of inhibition of bottlegourd mosaic virus. The degree of inhibition was found to increase with increase in the concentration of the surfactants, dilution of the virus and time of contact between the surfactants and the virus, suggesting a direct action of the surfactants on the virus.

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NOTES

- ¹ Verma (1973) reported that *Brassica oleracea* var. Worsing leaf extract inhibited TMV infection in *N. tabacum* var. Xanthi and *N. glutinosa*. The inhibitor was characterised as a polysaccharide. (*Indian Phytopath.* **26**: 713-722.)
- ² Varma (1968) reported that 0.05 M guanidine carbonate completely inhibited TNV infection in bean, possibly by blocking the formation of virus-induced RNA polymerase. (*Virology*, **36**: 305-308.) Guanidine carbamate and hydrochloride proved more effective in this than 8-azaguanidine and 2-thiouracil (Varma and Kamalesh Kumari, *HAU J. Res.*, **2**: 252-255 (1973)).
- ³ If alanine, serine and glutamic acid were applied before inoculation, rather than after inoculation, they inhibited TMV multiplication in tobacco more effectively. (Varma, J. P. and Verma, G. S. *Phytopath. Z.*, **58**: 53-58.)

9

Control

Control of plant virus diseases is a complicated problem. Firstly the plant viruses unlike other pathogens on infection harbour almost continuously and systemically in the whole plant system as long as the plant remains alive. Secondly, most of these maladies are actively spread in nature by the agency of insect vectors which breed on a large number of hosts. Some of these virus diseases are extremely contagious, affecting a large number of hosts belonging to several families and genera of plants. They are also carried through seed and vegetatively propagated plant parts like suckers, tubers and setts.

Several conventional methods are known for preventing or reducing the losses caused by virus diseases. Removal of diseased plants and 'volunteers' from the fields is probably the oldest method and usually first to be tried, e.g., in case of groundnut, volunteer plants carried 'bunchy top' and 'chlorosis' viruses during off season (Sharma, 1966).

Researches on these control measures have been directed primarily into two channels, namely, cultural practices and, heat and chemotherapy.

CULTURAL PRACTICES

The cultural practices include rotation of crops, roguing, fertiliser application, spacing, destruction of weeds, altering sowing dates, etc.

Infection can be controlled by roguing. This however, can be efficiently applied only in some virus diseases and specially where plants are young and the incidence of diseases as well as vectors is not high, e.g., potato virus diseases (Nagaich and Agrawal, 1969).

and virus diseases of cardamom (Varma and Capoor, 1953; Sharma *et al.*, 1972).

Rotation of crops may reduce the soil borne virus diseases. Remnant of crops of previous year also serve as an important source of virus infection. These and some volunteer plants such as potato and sugarbeet are liable to serve as reservoir of many viruses.

The practice of preparing seed beds in clean and isolated places may be helpful in raising seedlings free from infection with viruses. Effects of manuring on the disease incidence have little prospect in significantly controlling the virus diseases without affecting the crop yield. Generally, manuring for maximum yield also increases virus disease incidence. Somewhat similar results with regard to PVX indicated a direct relationship of the concentration of virus in the yield and tubers (Ganguly *et al.*, 1963). Further, iron seems to be a limiting factor for multiplication of the virus in tomato.

With regard to the vegetatively propagated crops such as potato, strawberry, raspberry, banana, cardamom, etc., it is very important to start with the virus-free crop.

The source of seed certification and also field certification has been very helpful in obtaining disease free seeds.

In India, seed certification of potatoes was initiated at IARI (Vasudeva and Azad, 1952; Vasudeva and Lal, 1949) and as a result, nucleus stock of potato varieties—'Darjeeling', 'Red Round', 'Kufri Chandramukhi', 'Kufri Sindhuri', 'Kufri Jyoti' and 'Up-to-date' were obtained. Incidentally, it may be mentioned that recent development in serological work enunciated by van Slogteren have also been found to be helpful in supplementing visual inspection required for enforcing such schemes and hasten the speed of testing (Nagaich *et al.*, 1969).

Many viruses are not commonly found in wild plants and are spread from cultivated crop to the next successively. The simplest way of controlling this type of spread is to break the cycle and have a gap between crops, but this will be successful only if the field is isolated or in an area where there are crop barriers. This method has been applied by Hopkins (1932) for tobacco leaf curl.

Lister (1960) has shown that some of the soil borne or nematode transmitted viruses, namely, arabis mosaic, tomato black ring, etc., are readily seed borne in several wild plants and it would

appear that this is their normal method of spread.

Budwood certification is practised for plantation crops like citrus, apple and others where spread of virus disease occurs through inadvertant propagation from infected stock. A long range citrus budwood certification programme for India has been recommended by Nariani and Raychaudhuri (1971). The cheapest way to avoid continuous increasing damage from virus diseases is to use tolerant varieties and combinations and, propagation stocks free of important viruses.

A comprehensive programme to establish virus indexed, virus free material in foundation blocks has to be raised in order to supply virus indexed budlings and stocks. Cross protection afforded by mild strains against virulent strains of viruses has been reported in many virus diseases. However, the only example of a large scale experiment on this method of control is in the swollen shoot disease of cacao. Posnette and Todd (1955) reported that out of 415 trees which had been infected with the mild strain of the virus, only 35 developed virulent symptoms later.

Several species of *Solanum* exhibit immunity to potato virus Y (Easton *et al.*, 1958) and X (Ross and Baerecke, 1950; Stelzner, 1950; Ross, 1958; Peterson and Hooker, 1959; Webb and Schultz, 1961). The varieties Corbett-Refugee of *Phaseolus vulgaris* has been shown to be immune to common bean mosaic virus (Walker and Jolivet, 1943).

One of the promising methods of controlling virus disease is to breed immune or resistant varieties of plants, e.g., chilli varieties 'Puri Red' and 'Puri Orange' *bhindi* (Lady's finger) variety 'Pusa Sawani', bean variety 'Kentucky Wonder', large cardamom variety, 'Sawney' and cowpea varieties 'Sawanney', 'Early Sugar Crowder' and 'Taylor', pea varieties 'Canners', 'Perfection', 'Horal', 'Hundred fold' and 'Little Marvel', have been shown to be resistant to mosaic, yellow vein mosaic, 'chirke' and mosaic viruses of these crops, respectively (Anand *et al.*, 1961; Joshi *et al.*, 1960; Capoor and Varma, 1956; Yaranguntaiah and Nariani, 1963; Raychaudhuri and Chatterjee, 1961; Sreenivasan and Nariani, 1965). Besides sources of resistance have been determined in case of papaya mosaic, *bhindi* yellow vein mosaic, tomato leaf curl and banana mosaic in *Carcia cauliflora*, *Abelmoscus manihot* var. *Pungens*, *Lycopersicon peruvianum* and *Musa balbisiana* respectively (Nariani and Seth, 1958; Capoor and Varma, 1961, 1968; Nariani and Vasudeva, 1963).

Ganguly *et al.* (1970) found that four varieties of wheat, namely, 'Ridley', F 4647, E 6003 and E 6831 are moderately resistant while four other varieties, namely, NP 717, NP 745, NP 803 and NP 809 are highly resistant to the wheat streak mosaic virus. Rice variety Pankhari 203 has been found to be resistant to tungro virus (John, 1968). Also, Paliwal and Raychaudhuri (1965) could locate sources of resistance to maize mosaic virus in ten exotic inbred lines of indigenous open pollinated varieties and the preliminary studies suggested that resistance is due to double recessive conditions of 'bb' gene at one locus.

Field testing of casava selections and hybrids have indicated that S-1310, S-1315, S-2380, H-97, H-43, H-86, H-165 and 226 are highly field resistant (Chacko and Thankappan, 1969). The mulberry varieties 'Ichinose' and 'Kairyonezumegaishi' of *Morus alba* and Oshimasho and Kosen of *M. latifolia* are resistant to the mulberry mosaic virus (Raychaudhuri *et al.*, 1965).

Many weeds are potential sources of viral infection to the cultivated crops. These wild hosts serve as a reservoir of many viruses of important crops like phloem necrosis of tea, leaf curl of tobacco, mosaic of chilli, etc. In Africa tomato spotted wilt virus is frequently carried to tomato and tobacco from infected weeds by its vector, *Frankliniella insularis* (Van der Plank and Anderson, 1944) and to pineapples in Hawaii by *Thrips tabaci* from the weed *Emilia sonchifolia*. Weeds also harbour symptomlessly some soil borne viruses, which later infect cultivated crops grown in the same soil. Cadman (1956) has shown that a ringspot virus in weeds infects raspberries causing leaf curl diseases. Similarly, white clover serves as a source of potato purple top roll in Simla hills (Nagaich and Giri, 1973). *Amaranthus viridis* carries brinjal mosaic virus in nature and acts as source of secondary infection (Sharma, 1969).

So far, some of the methods have been dealt with which are used to protect the plants from being infected with viruses. However, various other treatments have been suggested and also successfully employed for destroying the virus in the infected plants without having adverse effect on the host cells. Heat-therapy and chemo-therapy are two very useful methods of curing plant virus diseases.

HEAT THERAPY

Heat therapy is based on the principle of protein denaturing treatment, which produces non-specific protoplasmic destruction and has been found to be very useful in the case of virus affected plants and plant parts.

Kunkel (1941, 1943) made noteworthy contribution by employing heat-therapy for the first time for curing peach potted trees infected with peach yellows, little peach, red suture and rosette. He treated the potted trees at 34 to 36°C for two to four weeks. Later he showed (1941) that the virus causing aster yellows could also be destroyed by heating the host-plants such as periwinkle (*Vinca rosea*) and *Nicotiana rustica* which could survive the temperature of 40°C for two weeks. Raychaudhuri (1953) reported cure of bayberry yellows in periwinkle by employing the same technique. Since then a large number of virus diseases have been cured by heat-therapy. These viruses are both graft and insect transmissible.

Kassanis (1952, 1954) showed that even the plants infected with viruses with higher thermal inactivation end points could be cured by keeping them around 37°C.

There are certain examples of successful heat therapy of mechanically transmitted viruses as well. Hollings and Kassanis (1957) freed chrysanthemum plants from infection with aspermy, stunt and ring pattern viruses by exposing them at 30°C for three to four weeks.

A number of fruit trees, such as apple, cherry, peach, strawberry, raspberry, etc., have also been cured by heat-therapy. Grant (1957, 1958) obtained virus free budwood by treating Key lime infected with T₃ strain of tristeza virus at 98–104°F for 86–100 days. Greening affected budwood could be freed from the pathogen by hot moist air treatment at 47°C for four hours or 45°C for six hours (Nariani *et al*, 1972).

Considerable interest has been shown by growers in other countries in thermotherapy of certain crops such as strawberry, raspberry, chrysanthemum, carnation, sugarcane, citrus, pome and stone fruits, etc. Thirumalachar (1954) showed that potato tubers stored in thatched huts in Bihar for six months at average temperature between 29–36°C were freed from leaf roll, whereas others maintained in cold storage were all found to be infected. In the plains of India, the summer temperature shoots upto 40°C which

thus could be utilised with advantage (Nagaich and Upreti, 1964 and Upreti and Nagaich, 1968).

In India, hot air therapy was first tried against ratoon stunting disease of sugarcane from Madhya Pradesh (Singh, 1966). Grassy shoot disease is of virus origin and is primarily transmitted through seed pieces. Heat therapy was found to inactivate the virus in seed material. Treatment of infected seed pieces in water at 50°C for 2 to 2½ hours depending on the thickness of the cane and also keeping the seed canes in hot air at 54°C for eight hours in an airtight chamber found to inactivate the virus (Singh, 1968).

CHEMOTHERAPY

It is most desirable for the treatment of virus affected vegetatively propagated individual plants. Such treatment of the diseased material is essential for raising nucleus stock of virus free plants as in the case of potato, cardamom, strawberry, dolichos, mulberry, banana, etc. At Kalimpoing Plant Virus Research Station Raychaudhuri and Chatterjee (1961) found that soil drenching of 0.1 per cent thiouracil is effective in inhibiting the virus causing 'chirke' disease of large cardamom.

For the crops heavily infected with viruses even a little delay or modification in the severity of the symptoms by use of chemotherapeutic methods is sufficient for collecting the seeds or completing the breeding schedule. As a matter of fact, such reduction in the severity of symptoms of a number of virus diseases such as TMV (Nichols, 1952), potato leaf roll (Locke, 1948), tomato streak (Bhatt and Verma, 1958), tomato leaf curl (Lal and Singh, 1961), tomato stunt (Maramorosch, 1957) and tobacco leaf curl (Nariani, 1963) have been demonstrated by spraying growth regulators and related compounds on the diseased plants. Holmes (1955) prevented the death of tobacco plants, hypersensitive to TMV by watering 0.01 per cent thiouracil. A complete protection against systemic development of some viruses has been experimentally achieved by spraying or watering the young plants at an early stage of infection with such chemicals as 8-azaguanine and 2-thiouracil (Matthews, 1955; Raychaudhuri and Sharma, 1962).

RADIOTHERAPY

A number of viruses such as tobacco mosaic, tomato bushy stunt, *Cucumis* virus 2C and radish mosaic have been inactivated by irradiation (Price and Gowen, 1937; Bawden and Pirie, 1938; Raychaudhuri *et al.*, 1950; Raychaudhuri and Prasad, 1960). Rosberg (1959) observed that irradiated seeds of watermelon infected with tobacco ring spot virus reduced the incidence of the disease in the next crop.

ERADICATION OF VECTORS

A likely method of controlling the virus diseases which spread through insects is to prevent these insect vectors from entering into the field where the susceptible crops are grown. This can be achieved by screening the seedling beds. It is also possible to avoid insects by selecting the sites where they are not able to multiply actively due to unfavourable temperature, humidity or wind. Davis (1935, 1936) reported that *Myzus persicae*, the chief vector of potato viruses does not fly readily at a temperature below 18°C at a relative humidity above 75 per cent and during weather conditions without any sunshine. Similarly, in some tropical countries it is possible to raise healthy seed potatoes in areas where the temperature is too high for the aphids to exist (Porter, 1935; Bal and Norris, 1945). Since life-history of *Myzus persicae* is known it is possible to avoid these aphids by growing potato crop in areas where *M. persicae* cannot find facilities for over-wintering, or in seasons when aphid populations are low (Nagaich *et al.*, 1969).

Sometimes it has been possible to avoid infestation of vectors by early sowing of a crop such as sugarbeet. The condition of plant itself may also determine the degree of infestation of aphids. It has also been suggested that even the colour of the plant may play a part and the brown variety of lettuce is perhaps less frequently infected with mosaic than the green and yellow varieties. Self-sown plants of groundnut carried *Aphis craccivora*, the vector of chlorosis virus during off season (Sharma, 1966).

There have been many earlier reports claiming successful use of insecticides in controlling virus diseases (Watson, 1937; Magee *et al.*, 1942; Du Toit, 1948). The results were not very convincing.

However, Broadbent *et al.* (1956, 1958), tried several insecticides like D.D.T. endrin, sohradan, mipufax, malathion and systox and successfully prevented the spread of potato leaf roll virus from infected plants within the crop. In the case of non-persistent viruses, e.g., potato virus Y, these insecticides could neither protect the entry of the virus into the host nor stop its spread, though the incidence was decreased a little (Patkar *et al.*, 1969; Nirula and Kumar, 1967).

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10

Cereals

BARLEY (*Hordeum vulgare*)

Barley mosaic, Barley stripe mosaic and Barley yellow dwarf viruses are known to occur in tropics.

Mosaic

Symptoms: Affected plants remain stunted and become chlorotic. Late in the season typical mosaic symptoms develop on the affected leaves (Dhanraj and Raychaudhuri, 1969).

Transmission: The causal virus is readily transmitted mechanically as well as by the corn aphid, *Rhopalosiphum maidis*. The virus is seed borne (Dhanraj and Raychaudhuri, 1969).

Properties: The virus has thermal inactivation point between 53 to 55°C, the longevity *in vitro* of eight hours at room temperature (19–20°C) but becomes innocuous after ten hours. At low temperature the virus withstands storage for fifteen hours at 14 to 16°C.

Electron micrographic studies showed the virus particles to be spheres, the average diameter being 40 nm (Dhanraj and Raychaudhuri, 1969).

Host range: The disease is readily transmissible to barley, oat and wheat plants. The incubation period ranges from three to seven days. The virus is seed borne in barley varieties N.P. 13, N.P. 113, C. 138-2, K-71, K-24, E.164 × E.B. 533, C. 164 × E.B. 969, K12 × B.R. 32 and K251 out of 47 varieties tested. The seed transmission varies from 2 to 45 per cent.

Resistant varieties: All Mexican varieties tested exhibited a high degree of resistance. The varieties N.P. 13, N.P. 113, K 71, C 138-2 and N.P. 104 were, however, susceptible.

It has been recorded so far only from India.

Stripe Mosaic

Symptoms: The disease is characterised by development of necrotic somewhat irregular dark brown stripes that sometimes take the form of a 'V' or an inverted 'V'. The distal end of the affected leaves remains healthy while the proximal end develops a mosaic pattern or chlorosis.

Transmission: The virus is readily transmitted by mechanical inoculation and is seed and pollen borne. There is no information regarding vector.

Properties: The thermal inactivation point is 63°C, the dilution end point is 10^{-4} , longevity, *in vitro*, i.e., 13 days, in dried leaves, 95 to 98 days in frozen leaves at -10° (Ohmann-Krentzberg, 1962). The virus particles are rod-shaped and measure about 20 nm wide and in aggregated form are 135 to 175 nm long. Heating to 50°C causes the particles to aggregate linearly, when suspended in borate buffer at pH 7.5 and these are rendered inactive at pH below 4.5 (Kassanis and Slykhuis, 1959).

Host range: Sorghum vars. react to the virus with the development of local lesions only, while several species of wild and cultivated grasses are susceptible. The virus is found to be seed borne in 19 of the grasses tested (Nitzany and Gerechter, 1962). The virus is readily transmitted to barley, wheat, rye, maize, sorghum and a number of grasses. It also infects rice and oats including *Nicotiana tabacum* var. Samsun, *Chenopodium album* and *C. amaranticolor* (Singh *et al.*, 1960).

Control: Skimmed milk and whey reduced the disease spread (Hagborg and Chelack, 1960).

Geographical distribution: Australia, Europe, Israel, Japan, New Zealand and USA (Slykhuis, 1962).

Yellow Dwarf

Symptoms: Yellowing of the leaves, starting at the base of the lamina is the first visible symptom. The characteristic, golden yellowing of leaves together with stunting, are suggestive of the name of the disease.

Transmission: The disease could not be transmitted to barley and oat mechanically or through soil, however, aphid vectors, *Macrosiphum avenae*, *M. granarium*, *M. dirhodum*, *Rhopalosiphum*

maidis, *R. padi* and *Schizaphis granarium* transmitted the disease. **Host range:** Nagaich and Vashisth (1963) reported the occurrence of barley yellow dwarf from India and transmission of the disease to oats, barley, wheat, *Bromus* and *Lolium* seedlings.

Harpaz and Klein (1965) isolated barley yellow dwarf virus (BYDV) for the first time from naturally infected oats and observed that it is readily transmitted to Clinton oat seedlings by *R. padi* but not by *Macrosiphum granarium* or *M. dirhodum*.

Control: Treatment of barley with systemic and contact insecticides against the vectors *M. dirhodum*, *M. granarium*, *M. avenae* of barley dwarf virus sometimes delayed infection but eventually all the plants become infected (Dickason *et al*, 1960). A total of 6,728 barley varieties were inoculated with yellow dwarf virus by means of viruliferous aphids using dwarfing as the most reliable criterion of infection. Twenty-eight varieties were rated highly resistant, thirty-four resistant and fifty-five fairly tolerant.

The disease is found to occur in North America, Europe and Australia (Bruchl, 1961).

MAIZE (*Zea mays*)

Dwarf Mosaic

The disease was first observed in 1962 in Ohio (USA) by Janson and Ellet (1963). In the following years it was also observed to be present in other corn growing areas resulting in huge losses.

Symptoms: The symptoms develop as diffused mosaic mottling of the young leaves. The plants when infected early, remain very much stunted with poorly developed ears.

Transmission: The virus is sap transmissible and symptoms in maize seedlings take about one to two weeks to develop. The virus transmitted by 12 species of aphids (Granados, 1969), is stylet borne. *Dactynotus* sp, is more efficient vector (over 60 per cent transmission) and *Rhopalosiphum maidis* is less efficient (under 10 per cent transmission).

Properties: The properties of maize dwarf mosaic virus are generally similar to those of sugarcane mosaic virus to which it is serologically related. The thermal inactivation point is 53 to 55°C, the dilution end-point is 1:1000 (Adsuar, 1950) and the longevity *in vitro* is two to twenty four hours (Rafay, 1935).

The virus particles are uniform and average about 773 nm in

length. The purified virus contains 5.6 per cent nucleic acid and the protein, nucleic acid ratio is similar to that of tobacco mosaic virus (Schgal and Jean, 1970).

Host range: Amongst its hosts are, sugarcane, maize, sorghum, sudan grass and Johnson grass. The antiserum of the virus reacted weakly with sugarcane mosaic virus. The virus has been identified as a strain of sugarcane mosaic virus (Williams *et al.*, 1965).

Mosaic

Symptoms: The symptoms in the infected plants are variable, owing to the intensity of infection, i.e., of mosaic mottling. The symptoms appear in the infected plant at the base of the young unfolding leaf which is rendered small in size and pale green in appearance. The cobs borne by diseased plants are usually not well filled.

The disease can cause a reduction in yield to the extent of 31.81 per cent. Potassium deficiency increases the effect of the virus on the crop yield (Raychaudhuri *et al.*, 1968).

Transmission: The virus is transmitted by several species of aphids, namely, *Aphis maidis*, *A. gossypii* and *Macrosiphum granarium*. The last two are more efficient vectors. Bhargava and Shukla (1966) reported *Myzus persicae* as the vector. The virus is sap transmissible and is not transmitted through seed (Chona and Seth, 1960).

Properties: Epidermal cells of infected maize leaves show round or longitudinal inclusion bodies, mostly attached to the nucleus or lying close to it. After giemsa staining they appear pink to purple. The round bodies are 2–7.7 nm in diameter, while the longitudinal ones are 3–4.6 × 14–26 nm (Paliwal and Raychaudhuri, 1965). The virus is inactivated at a temperature higher than 55°C by dilution beyond 1:100 and by storage for more than sixteen hours at room temperature (28–32°C).

Host range: The host range of the virus includes maize (*Zea mays*) and its several varieties. *Sorghum vulgare* Pers., *S. sudanense* (Piper) Stapf., *Dactyloctenium aegyptium* Beauv., *Coix lachrymajobi*, *Euchlaena mexicana*, *Digitaria bifasciculata*, *Setaria verticillata*, and *Brachiara ramosa* and also *Phalaris minor*, a winter grass as a symptomless carrier (Chona and Seth, 1960).

On the basis of biological and physical properties, host range and cross-protection tests, it is considered as a strain of sugarcane

mosaic virus (Seth and Raychaudhuri, 1967).

Control: Some exotic inbred lines have been found to be highly resistant.

It has been reported from India

Rough Dwarf

Symptoms: The disease is characterised by dwarfing in early stage of growth of the crop and the leaves at the apex are chlorotic, reddened and often withered at the tips. Numerous 'galls' appear on the leaf veins as white pustules and thus appear rough in texture. The root system of affected plants is greatly reduced, discoloured and rotted at the extremity.

Severely affected plants remain sterile, the male inflorescence emerges with difficulty and the female inflorescence remains rudimentary (Biraghi, 1952).

Transmission: The virus is transmitted by two species of leaf-hoppers, namely, *Laodelphax striatellus* and *Javesolla pellucida* the latter being the efficient vector (Harpaz *et al.*, 1965).

Properties: The thermal inactivation point of virus lies between 60° to 80°C. The longevity *in vitro* is found to be one month. The virus particles are isometric, measuring about 60 nm in diameter. Some particles show an internal core approximately 34 nm in diameter (Lovisolo *et al.*, 1967).

Host range: The virus infects *Hordeum vulgare*, *Avena sativa*, *Triticum vulgare*, *Sorghum vulgare* and *Oryza sativa* (Vidano *et al.*, 1966).

It has been reported from Israel.

Streak

Symptom: The disease is characterised by production of chlorosis in the affected leaves confined to broken stripes arranged along the veins.

Transmission: The maize streak virus is not carried in the seed, neither transmitted by any mechanical method of inoculation. The insect vectors are three species of leaf hoppers, *Cicadulina mbila*, *C. zeae* and *C. nicholsi*. Of these the first one is the most efficient vector.

Host range: It is recognised that wild grasses serve in harbouring maize streak virus. The two grasses, namely, *Digitaria horizontalis* and *Eleusine indica* are commonly affected.

Control: Resistance to maize streak virus is mainly controlled by a major gene, with some minor genes acting as modifiers. This resistance was transferred to 36 desired lines from 6 susceptible maize groups (Storey and Hawland, 1967a).

It has been reported from Africa and Egypt.

Vein Enation

Symptoms: The disease is characterised by severe swelling of the veins on the lower side of leaf lamina. Numerous elongated enations or galls develop on the leaf veins. Affected plants are stunted with chlorotic leaves and many of them killed prematurely. Leaves of affected plants showed twisting habits. Tassels of diseased plants rarely emerge out and female inflorescence is either poorly developed or is rudimentary.

Transmission: The disease is not transmissible by sap or seed but is transmitted by a jassid vector, *Cicadulina intolita*. Both nymphs or adults are able to transmit the disease. A single viruliferous adult or nymph can transmit the virus. The insects could acquire the virus after a feeding of 20–30 minutes on diseased plants. Incubation period of the virus in its vector is about twenty hours.

Host range: The virus is transmissible to wheat, rice, sugarcane, ragi, sorghum, oat, *Setaria glauca*, *Eleusine indica*, *Paspalum sanguinale* and *Coix lachryma-johi* (Ahlawat and Raychaudhuri, 1975).

Distribution: The disease is prevalent in Darjeeling hills (India).

OAT (*Avena sativa*)

Red Leaf

The disease is caused by cereal yellow dwarf virus.

Symptoms: The disease is identified by development of pale yellow blotches on leaf tips. Later on due to the fusion of the blotches the leaf becomes reddish brown. In advanced stages the entire lamina becomes discoloured and plants are generally stunted and bushy in appearance. The leaves of the affected plants are stiff and erect. Blasting of basal florets is common (Nagaich and Vashisth, 1963).

Transmission: The virus is transmitted by *Rhopalosiphum* spp. (Vashisth, 1967).

Host range: Varieties of oat, Kent Algerian, Khesari, Barley

millar, Nip and two selections FOL 29 and W. 11 were found to be susceptible. It has been reported from India.

RICE (*Oryza sativa*)

A number of viruses are known to infect rice in tropical countries. Some of the viruses are widespread and are known to cause heavy losses.

Dwarf

Symptoms: The disease is characterised by the presence of chlorotic and broken yellowish white streaks parallel to the veins on the leaves. Sometimes older leaves of infected plants show diffuse yellowing on the distal end and along the margin. The entire plant is stunted and usually produces many diminutive tillers and the number of tillers may also be reduced. Panicles, if produced are poor, carrying small number of grains many of which remain empty. The grains are seen to be covered by dark brown blotches.

Transmission: *Nephotettix cincticeps*, *N. nigropictus* and *Recilia* (*Inazuma*) *dorsalis* have long been known to be the vectors of the virus (Ou, 1972; Nasu, 1963).

Transmission of rice dwarf virus has been studied in detail by Fukushi (1933, 1940) and later by Shinkai (1962) and Nasu (1963). The presence of the virus was demonstrated in the egg. **Properties:** Electron microscopy of purified virus preparations showed icosahedral particles of 70 nm in diameter (Fukushi *et al.*, 1960). Infectivity is destroyed by storing the virus extract for seventy-two hours at 0–4°C. Virus in frozen diseased leaves and viruliferous leaf hoppers withstood 30 to 35°C for five months. The thermal inactivation point is 40–45°C (Fukushi and Kimura, 1959; Kimura and Fukushi, 1960).

The disease has been reported from Japan and the Philippines, probably Korea and China. The identity of the 'dwarf' and 'stunt' disease reported from the Philippines (Agati *et al.*, 1941; Reyes, 1957; Reyes *et al.*, 1959; Serrano 1957) is uncertain but the records probably refer to tungro and other diseases (Ou and Ling, 1966).

Host range: It is transmitted to wheat, oat and weeds, namely, *Echinochloa crusgalli* and *Paspalum thuberosum* (Fukushi, 1934).

Rice Grassy Stunt

The disease was first reported in Philippines (Rivera *et al.*, 1966) and later on from Ceylon and Thailand. It is suspected to be present in India (Raychaudhuri *et al.*, 1967).

Symptoms: Characteristic symptoms of the disease are severe stunting with profuse tillering. Leaves become short, narrow, erect, having numerous rusty spots or blotches. Profuse tillering sometimes gives a fan like rosette appearance to the plant.

Transmission. The disease is found to be transmitted by brown plant hopper, *Nilaparvata lugens* (Rivera *et al.*, 1966). The incubation period in the insect varies from five to twenty-eight days while that in plant from ten to twenty days. Sogawa (1973) made some detailed studies on the feeding behaviour of the vector and the varietal resistance of rice to this insect. In course of stylet penetration the insect ejects a coagulable salivary secretion when rapidly set to form gel, thus encasing the stylet and forming the 'stylet sheath' within the plant tissue. After insertion of stylet into the vascular tissue, the insect ceases to secrete the saliva and starts sucking sap.

The causal organism of the disease, measuring 70 nm in diameter, was observed in cells of the insect vector (IRRI, 1968). Earlier, it was regarded to be a virus disease (IRRI, 1967). According to Shikata (personal communication) the disease could not be cured by application of antibiotics for about two years. No MLO has yet been reported from infected plant tissue.

Hoja-Blanca

Symptoms: The characteristic symptoms appear as white chlorotic stripes on the leaves or completely white leaves.

Transmission: The virus is not transmitted by sap inoculation. *Sogatia oryzicola* is known to be principal vector. The disease is also transmitted by *S. cubans* which is a minor vector. Galvez *et al.* (1960) transmitted the virus using *S. cubans* from rice to rice or from *Echinochloa colonum* to rice. Such transmission has been shown to be possible by forced feeding with highly active individuals of *S. cubans* (Galvez, 1968) *Sogatia oryzicola* transmits the virus with varying results depending on the age of the rice plant and the point of inoculation. The shortest acquisition feeding period is fifteen minutes and incubation period ranged from five to thirty-seven days. The shortest inoculation feeding period is

thirty minutes and incubation of plants varies from three to forty-five days, according to the age of the seedling (Galvez, 1968, 1969). The existence of transtadial and transovarial passage has been proved by several workers (Galvez, 1969).

Properties: The virus particles are spherical and measure 42 nm in diameter (Herold *et al.*, 1968). Shikata and Galvez (1969) have observed thread-like particles in the vector and infected plants. It has been observed from North, Central and South America.

Host range: The virus is transmitted to *Echinochloa colonum*, *Leptochloa* sp., *Digitaria* sp., *Triticum sativum*, *Hordeum vulgare* and *Avena sativa* (Galvez *et al.*, 1960, 1961). Lamey *et al.* (1961, 1964) also transmitted the virus to rye (*Secale cereale*) barley, oats and wheat.

Mosaic

Symptoms: The infected plants exhibit foliar mottling. These mottled areas are irregular in shape and vary in size. Tillering is reduced. Severely infected plants are stunted and the leaves turn yellowish brown and eventually wither.

Transmission: The virus is transmitted by mechanical means such as rubbing with carborundum and inoculation by pin prick method. Disease is transmitted from rice to maize, but not from rice to rice.

It is suspected that the mosaic disease of rice is similar to the mosaic disease diagnosed on other graminaceous plants (Martinez *et al.*, 1960). It has been reported from the Philippines.

Orange Leaf

Symptoms: The disease is identified by orange discolouration of the leaves starting from the tip. The number of tillers is reduced but there is no conspicuous stunting. Rapid death of seedlings occur. At high temperature (30°C) the disease is more prevalent and plants die sooner.

Transmission: Rivera *et al.* (1963) showed that the virus is transmitted by leaf hopper *Recilia dorsalis* (Motscha).

Host range: Glasshouse trials with a limited number of rice varieties indicated that Peta, FB-121 and others are resistant while susceptible varieties include Tjerimas and BP 1-76. It has been reported from Philippines, Thailand and Ceylon.

Yellow Mottle

Symptoms: The symptoms include stunting, reduction in the tillering and crinkling, mottling, yellow streaking of the leaves, malformation and partial emergence of the panicles, along with sterility in severe cases (Baker, 1970).

Transmission: The beetle *Sleselia pusilla* transmits the virus. The virus is stable and highly infective and transmitted mechanically.

Host range: It is transmitted to several varieties of *Oryza sativa* and *O. punctata* but not to *O. sichingen*, barley, *Pennisetum typhoides*, durum wheat, maize, oats, rye, sorghum, wheat, sugarcane and some grasses (Baker, 1970). It has been reported from Kenya.

Transitory Yellowing

Symptoms: The characteristic symptoms are leaf discolouration, reduced tillering and stunting. Infected plants bear few panicles or none. The discolouration appears on the two lower leaves which become distinctly yellow and finally turn bright yellow or orange buff according to the severity of the disease. Finally, the diseased leaves fall off. Following the typical appearance of leaf yellowing, infected plants recover gradually and produce no symptoms during the later stages of the growth and frequently, healthy looking tillers grow from a diseased stalk. For this reason the name 'transitory yellowing' is given to the disease. Large, round inclusion bodies are found internally in the parenchyma cells, sieve tubes, etc., around vascular bundles (Su and Huang, 1965).

Transmission: Chiu *et al.* (1965) first observed *Nephotettix nigropictus* as the vector; later *N. cincticeps* was also found to be a vector of the virus (Chiu and Jean, 1969; Chiu *et al.*, 1968). It is reported from Taiwan.

Tungro

Tungro which means degenerated growth, was first observed in the Philippines in 1963 and its viral nature was confirmed by Rivera and Ou (1965). Tungro is distributed throughout South East Asia and known by different names mostly based on symptom expression but the investigators of different countries could not confirm the disease as tungro. 'Penyakit Merah' (Red disease) was known to occur in Malaysia since 1938 and was identified as tungro in 1965 by Ou. 'Mentek' (Midget) disease of Indonesia is known since

1859, but the similarities in symptomatology, varietal reactions, vector-virus interaction and other circumstantial evidences indicate that it is also same as tungro (Ou, 1965; Rivera *et al.*, 1968). According to Wathanakul and Weerapat (1969) Wathanakul in 1964 identified yellow orange leaf as a distinct disease of rice. Later on Lamey *et al.* (1967a) concluded that yellow-orange leaf disease was similar to tungro. The tungro was also first reported from India by Raychaudhuri *et al.* (1967) which was same as shown by John (1966). In addition to the distribution of tungro in the Philippines, India, Malaysia, Thailand and Indonesia, tungro has also been reported to be occurring in East Pakistan (Galvez and Miah, 1969; Lippopold *et al.*, 1970).

Tungro is one of the most destructive rice diseases in tropical countries. During 1934-36, 'Mentek' disease affected 30,000 to 50,000 hectares of paddy cultivation in Java province of Indonesia (van der Vecht, 1953). In Thailand tungro damaged half of the 66,900 hectares in 1966. An outbreak of tungro in 1971 affected severely the crop on large areas in the Philippines. In the experiments conducted at IARI, New Delhi, tungro infection, on an average, in a standing crop of TN 1 causes 75 loss in grain weight. Recently, tungro epidemic was also observed in Eastern Uttar Pradesh and North Bihar, India (John, 1970) and again in Indonesia during 1972. In recent years due to the cultivation of resistant varieties the disease occurs in the pockets where old susceptible varieties are still in practice (Rivera *et al.*, 1968). Some such areas are reported to be in existence in West Bengal (Lowe, 1972). **Symptoms:** Tungro affected rice plants, are stunted and have reduced number of tillers. The young emerging leaves develop interveinal chlorosis leading to discolouration of the leaves, starting from tip downwards. Often whole leaf is discoloured. The leaf discolouration is in various shades of yellow, common in Japonica varieties but in Indica varieties orange colouration is common. Root development is poor. Plants infected at an early stage generally die prematurely. Infected plants take more time for maturity because of delayed flowering. The panicles are often poorly developed, small and sterile. The grains are often covered with dark brown blotches and are lighter than those of healthy plants.

Transmission: Seed transmission of tungro virus always gave negative results (Singh, 1969). Similarly no evidence has been

found to indicate its soil or mechanical transmission.

The virus is, however, transmitted by *Nephotettix virescens* (*Nephotettix impicticeps*) previously known as *N. nigropictus* (Ishihara, 1964). Later, (IRRI, 1968, 1969) *N. nigropictus* (*N. apicalis*, *N. narvus*, *N. malayanes*, Ling, 1970) and *Recilis dorsalis* were also found to transmit the virus though low in percentage. Negative transmission of the disease by *N. nigropictus* has been obtained by various workers (John, 1968; Lamey *et al.*, 1967b; Ling, 1968; Ou and Rivera, 1969; Singh, 1969).

About 83 per cent of *N. virescens* in a population are active transmitters (Rivera and Ou, 1965). Singh (1969), however, reported an average of 35 per cent as active transmitters. In case of *N. nigropictus* 27 per cent are active transmitters (Ling, 1970), whereas 4 per cent (IRRI, 1968) to 8 per cent of *Ricilia dorsalis* transmit the virus (Rivera *et al.*, 1969).

The shortest acquisition feeding period for *N. virescens* is five minutes as reported by Singh (1969). Others have reported thirty minutes (Rivera and Ou, 1965; John, 1968; Lim, 1969) as the optimum acquisition feeding period. Ling (1966) found very unusual virus vector interaction. There is no apparent incubation period in the insect, this being an example of a non-persistent leaf hopper borne virus. According to Ling (1966), the incubation period cannot be longer than two hours because a virus-free *N. virescens* can transmit the disease by having acquisition and inoculation feeding period of one hour each. The insects, however, do not retain the virus for more than five to six days. Viruliferous insects usually transmit the disease immediately after their acquisition feeding and continue to transmit every day until they lose their infectivity. Once they lose their infectivity they remain non-infective for the rest of their life. The longest retention period for *N. nigropictus* is three days (Ling, 1970) and for *R. dorsalis* is four days (Rivera *et al.*, 1969). Nymphs are as good transmitters as adults but there is no transtadial or transovarial passage (Ling, 1966).

The shortest inoculation feeding period varies from seven minutes (Ling, 1968a) to thirty minutes (Lim, 1969). The incubation period in plant varies from five days (Rivera and Ou, 1965) to fifteen days (Wathanakul and Weerapat, 1969).

The virus seems to have no deleterious effect on *N. virescens* because of no significant differences in life span, fecundity and

rate of hatching between viruliferous and non-viruliferous insects (Ling, 1968a).

Properties: Ou and Ling (1967) published the work of Shikata who took the first electron micrograph of tungro virus in ultrathin sections of diseased leaf and measured the particle size which is 30–35 nm in diameter. Galves (1968) purified the virus and reported that the particles are polyhedral with a diameter of 30–33 nm.

Galvez (1968) studied the physicochemical characteristics of the virus by analytical density gradient centrifugation. The virus withstands temperatures below 63°C for 10 minutes and pH value upto 9 without apparent denaturation. The virus can be kept at room temperature (*in vitro*) for more than twenty-four hours. The sedimentation coefficient is $175 \pm 5S$.

Antiserum to the tungro virus has been obtained by injecting partially purified material into rabbits. The antiserum, however, is non-specific, i.e., not only to tungro virus but also to orange leaf, yellow dwarf and grassy stunt (John, 1965).

Strains of tungro: The tungro virus is known to have three strains. Rivera and Ou (1967) reported 'S' and 'M' strains of the virus. Although, both the strains produce similar symptoms on varieties like TN-1, IR-8 and Tainan-3, they can be differentiated by their reactions on rice varieties, namely, Acheh, FK-135, Pacita. The 'S' strain in these three varieties produces conspicuous interveinal chlorosis, giving an appearance of yellow stripe and sometimes irregular chlorotic specks on younger leaves. On the other hand, the 'M' strain produces only mottling. The 'S' strain is widely distributed in the Philippines and also in India. Anjenayulu and John (1972) further divided the 'S' strain into four substrains depending on the reactions on some more differentials. The strain vary in their virulence on the variety, TN-1 and appear to be distributed in different regions of India.

Recently another strain was described from the Philippines which has been designated as 'T' strain. The new strain incites narrow leaf blades on TN-1, IR-5, IR-8, IR-22 but produces interveinal stripes on FK-135 which closely resemble the symptoms produced by 'S' strain. The 'T' strain, however, retards growth less than 'S' strain, and 'M' strain (IRRI, 1970).

Host range: Raychaudhuri *et al.* (1967) reported that in addition to leaf yellowing, chlorosis has been observed on some grasses such as *Leersia hexandra*, *Rottboellia compressa* and

Cynodon dactylon occurring in rice fields.

Wathanakul (1964) inoculated twenty-nine species of grass weeds and found that *Eleusine indica*, *Echinochloa colonum* and *E. crusgalli* Beauv. are alternative hosts of tungro virus. Infected *E. indica* shows stunting and some leaf discolouration. No symptoms were observed on other hosts but virus was recovered from the inoculated plants. Rivera *et al.* (1969) reported that among sixty-three species of graminaceous plants inoculated, the following species were infected (all had less than six per cent transmission except species of *Oryza*). These are *Dactyloctenium aegyptium*, *Eragrostis tenella*, *Ishaenum rugoalum*, *Leersia hexandra* and *Oryza barthii*.

Recently Mishra *et al.* (1973) tested some common grass hosts, surrounding paddy fields, and confirmed five hosts to harbour the virus in nature, namely, *Hemarthria compressa*, *Sorghum halapense*, *Polypogon monspoliensis*, *Eleusine indica* and *Sporobolus tremulus* and the transmission from these natural hosts ranged from 8 to 43.7 per cent. They concluded that these hosts harbour the virus in the off-season when paddy crop is not there and thereby help in perpetuation of the virus inoculum for the next crop. Besides these hosts, few other species of grassy hosts were artificially inoculated in the glasshouse which on back transmission gave positive results. These are *Setaria verticillata*, *Bothriochloa odorata*, *Pennisetum typhoides*, *Brachiaria raptans*, *Digitaria adacendens*, *Paspalum distichum*, *Dactyloctenium aegyptium*.

Varietal resistance: Based on the results of artificial inoculation the following are some of the varieties resistant to tungro virus. Katariabogh, Latisail, Tilakkachari, Pankhari 203 (reported from India), Dara, Peta (reported from Indonesia), Badshabogh, T. 412, Basmati 37, Bangwan, Fadjar, Indoasail, Gampai, Lantijang, M-sung song, Ram Tulasi, Raja Mandal Barain, Seri Raja, Tjeremma (from the Philippines), etc. Pankhari 203 is not only resistant to tungro but also to its vectors. Pankhari 203 and Latisail have been used as resistant donors by breeders at IRRI (Ling and Aguiro, 1970) and AICRIP, India.

Misidentified Diseases, Synonyms and Suspected Virus Diseases of Rice in Tropical Regions

1. *Leaf gall* (Philippines) transmitted by *Cicadulina bipunctata* was shown actually due to insect toxin (Maramorosch, *et al.*, 1961).

2. *Dwarf, stunt or a Accep na pula* (Philippines) transmitted by *Nephotettix bipunctatus* (Agati *et al.*, 1941), *N. bipunctatus cincticeps* (Serrano, 1957), *N. apicalis cincticeps* (Reyes, 1957; Reyes *et al.*, 1959). Based on symptomatology, species of vector and vector interaction, the dwarf disease reported in the Philippines is not identical with the dwarf disease recorded in Japan.

Rice rosette (Philippines) transmitted by *Nilaparvata lugens* (Bergonia *et al.*, 1966) appears similar to rice stunt in symptomatology, species of vector and virus vector interaction.

WHEAT (*Triticum vulgare*)

Many virus diseases of wheat have been reported from subtropical, tropical and temperate regions. Of these wheat mosaic, soil borne wheat mosaic, streak mosaic and wheat mosaic streak viruses are important in tropical regions.

Mosaic Streak

The mosaic streak of wheat caused by the 'Chirke' virus of large cardamom (*Amomum subulatum*) was reported by Raychaudhuri and Chatterjee (1958).

Symptoms: The affected areas show irregular streaks on the leaf. Gradually the streaks coalesce and eventually turn brownish and dry up (Ganguly *et al.*, 1970). The diseased plants are neither malformed nor reduced in size. Substantial reduction in the yield is caused by the virus.

Transmission: The virus is sap transmissible and also spreads through the agency of aphids, *Rhopalosiphum maidis* (Raychaudhuri and Ganguly, 1965), *R. padi*, *Brachycaudus helichrysi* (Raychaudhuri and Chatterjee, 1965) and *Sitobion avenae* (Raychaudhuri and Ganguly, 1968b).

Properties: The virus has been purified by differential centrifugation and viral particles are polyhedral with an average diameter of 40 nm (Raychaudhuri *et al.*, 1969). The virus is inactivated at 60°C and loses infectivity at dilution of 1:5000 and by storage at room temperature for eight days (Raychaudhuri and Chatterjee, 1964).

Host range: Large cardamom (*Amomum subulatum*), ginger (*Zingiber officinale*), an orchid (*Dendrobium gigantia*) and a perennial weed (*Acorus calamus*) are the alternative hosts of the virus

(Raychaudhuri and Ganguly, 1965, 1968a and 1968c). It has been reported from India.

Control: The reactions of 340 wheat varieties (*Triticum aestivum*) to the mosaic streak virus were recorded under artificial inoculation and natural field conditions. Four varieties, namely, 'Ridley', E 4647, E 6003 and E 6831 were resistant whereas 'NP 717', 'NP 705', 'NP 803' and 'NP 809' showed moderate resistance to disease (Ganguly *et al.*, 1970), 'Kalyan Sona', 'Sharbati Sonora', 'Pusa lerma', 'Safed lerma', 'Lerma Rojo' and 'Sonora 64' showed very severe reaction to the virus.

Mosaic

Symptoms: The mosaic pattern induced by the virus on the leaves of the affected plants is not well defined. The mottling is rather late on the wheat plants and consists of irregular streaks which vary in length and width. The greater portion of the infected leaves is of light green colour and shows up as dark green pattern on light green background.

Transmission: The virus is soil transmitted and it can remain infective in the soil as long as six years. The virus could not be mechanically transmitted by usual leaf inoculation methods or by the vector of wheat striate mosaic virus. A fungus, *Polymyxa graminis* is the vector. Rao and Brakke (1970) found that keeping plants in darkness and removal of the leaves of inoculated plants substantially increased the percentage of infected plants that showed visible symptoms. It has not been reported from India.

Properties: The virus has rod shaped particles with an average length of 160–300 nm, and 28nm in diameter (Herbert and Coleman, 1955).

Host range: It is transmissible to members of the Hordeae and the following species have been found susceptible to the infection *Triticum vulgare*, *T. compactum*, *T. turgidum*, *T. durum*, *T. dicoccum*, *T. spelta*, *T. polonium*, *T. monococcum*, *Hordeum aatrium*, and *Secale cereale* (Mckinney, 1930) also *Poa cinnure*.

Striate Mosaic

Symptoms: The infected plants are identified in the early stage of the presence of faint chlorotic dashes or streaks along the veins. Later these become visible most clearly on the lower surface of the leaves. Necrosis usually follows chlorosis, and leaf injury often becomes severe. Infected plants are stunted and yield partially

sterile heads and poor quality grain. Some plant die before the heads are formed.

Transmission: The disease is not transmitted through sap. The virus has leaf hopper *Endria inimica* as vector (Slykhuys, 1953).

Properties: Under electron microscope the virus particles have been observed to be bullet shaped and of about 65 nm in diameter and 270 nm in length.

Host range: The virus infects all common spring wheats. In addition *Triticum durum* and *T. dicoccum* have been infected experimentally. Barley, oats, *Eragrostis cilianensis* and *Panicum capillare* contract infection.

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Fibre Crops

ABACA (*Musa textilis*)

Bunchy Top

Symptoms: The disease symptoms include reduction in leaf size and narrowing of the blades; irregular and more rapid unfurling of young rolled leaves; darkening of the green colour of the leaf sheath and short results in rosetting at the crown of the plant and degeneration of the root system (Wardlaw, 1935).

Transmission: The virus is not transmitted by mechanical methods of inoculation. The insect vector is the aphid, *Pentalonia nigronervosa*.

Host range: The virus is restricted to *Musa textilis* and its different varieties.

Control: The disease which has been reported from the Philippines can be checked by using virus free suckers and also by inspecting young plantations regularly and roguing out the diseased plants (Ocfemia, 1930).

Mosaic

Symptoms: Mosaic symptoms in abaca consist of light coloured dot like areas 1/2 mm in diameter, on the surface of the leaf. In the beginning mosaic symptoms appear as pale-yellow to light green streaks running parallel to the veins between normal green tissues. The symptoms may occur on the leaf-blade, mid-rib, petiole, pseudostem, inflorescence and fruit. In the late stages the yellowish areas become rusty to dark brown. The symptoms appear to vary from variety to variety and among the strains of virus on any one variety (Kent, 1954).

Transmission: *Aphis gossypii* and *Rhopalosiphum nymphaeae* transmitted abaca mosaic in the Philippines. *A. maidis* further transmitted abaca mosaic to maize (Celino and Ocfemia, 1941). The virus is transmitted to healthy abaca, arrow root, maize and *Canna indica* seedlings by cutting a partly emerged infected leaf with a safety razor blade and then drawing the edge of the leaf to and from gently against the leaf surface (Celine and Martinez, 1956). The virus is also sap transmissible (Banigo *et al.*, 1965).
Host range: *A. gossypii* can transmit the virus from diseased to healthy *M. textilis* plants; from *Canna indica* to healthy *M. textilis*, *C. edulis* and ornamental canna varieties and, from maize to maize; *Rhopalosiphum nymphaeae* and *R. prunifoliae* from *M. textilis* to the same host and, *A. maidis* from maize to *M. textilis* transmit reciprocally and from *C. indica* to *M. textilis* (Ocfemia, 1949, 1953).

COTTON (*Gossypium* sp.)

Anthocyanosis

Symptoms: The disease is characterised by the presence of abnormally large quantities of purple or reddish pigments in the lower or middle leaves (Costa, 1956).

Transmission: The virus is graft transmissible. The vector is the aphid *Aphis gossypii*.

Host range: The virus infects *Gossypium barbadense*, *Hibiscus esculentum*, *Sida rhombifolia* and *S. microntha* (Costa, 1956).

In Brazil the above syndrome is also caused by magnesium deficiency and virus infection does bring about a reduction in the magnesium content of the foliage from 0.44 to 1.31 to between 0.61 and 0.31 per cent (Costa, 1956; Costa and Carvalho, 1962).

Leaf Crumple

Symptoms: The disease is characterised by the development of interveinal leaf tissues which undergo hypertrophy resulting in an inverted cupping of the entire leaf. The veins of severely affected leaves may become shortened. The hypertrophied surface is rough due to confinement by the veinlets. Vein clearing and interveinal chlorosis are observed in some leaves. A similar hypertrophy may also occur in petals.

Diseased plants also show typical crumpling of leaves and

remain stunted and flower sparingly (Costa, 1956).

Transmission: The virus is not sap transmissible but can be transmitted readily by two species of white flies, *Bemisia inconspicua* and *Trialeurodes abutilonia*. There is no evidence of seed transmission (Dickson and Laird, 1960). The disease has been reported from Brazil and South-eastern California, USA.

Leaf Curl

Symptoms: Apart from distinctive symptoms on the petals, the disease is characterised by occasional thickening of veins (Nair *et al.*, 1964). A leaf that is already fully grown never develops symptoms, but a partly grown leaf may develop thickening of the lower surface of the smaller veins. Such thickening starts at a number of points which gradually tend to join up, until all the veins are affected. Frequently the symptoms first appear on the very young leaves, the older grown ones developing normally. When an older plant that already has flower buds contracts the disease, it is often the epicalyx of the buds that show the first sign of thickening of the veins. The new leaves produced are small, exceedingly crinkled and curled at the edges, either upward or downward. The primary stems of the plant often grow taller than normal, the internodes being elongated and irregularly curved; but sometimes the whole plant is stunted in growth.

Transmission: The insect vectors are *Bemisia gossypiperda*, *B. goldingi* and *B. tabaci* (Nair *et al.*, 1964; Yassin and El Nur, 1970). The virus is neither sap inoculable nor it is carried in the seed or soil.

Host range: The virus infects hosts *Gossypium peruvianum* (Sakel cotton) *Hibiscus esculentus*, *H. aubdariffa*, *H. cannabinus* and *Althea rosea*.

Control: Two types of sakel cotton, namely X 1530 and X 1750 have proved extremely resistant to the virus under field conditions in Sudan (Bailey, 1934).

Leaf Mottle

Symptoms: The disease is characterised by an irregular mottling particularly noticeable against the light and most pronounced near the veins. Severely infected young leaves appear pale. The lobes of severely diseased leaves are often seen distorted and elongated and on many infected plants the main stem becomes

stunted. Flowering appears to be much suppressed.

Transmission: The disease is graft transmissible.

Host range: Virus infects Egyptian cotton XL₁, X_{1730A} and Domains Sakel in Sudan (Nair, 1959).

Veinal Mosaic

Symptoms: Symptoms consist of stunting, shortening of the internodes (bunchy top), a dark green coloration of the leaves, generalized or broken mottling of the veins, rugosity of the laminae and curling of the margins.

Transmission: The virus is easily transmissible by grafting.

Host range: It infects four species of *Gossypium*, *G. hirsutum*, *G. barbadense*, *G. punctatum* and *G. klotzchiarum*, some interspecific hybrids and *G. trilobum* (Costa and Foster, 1938), in South America.

JUTE (*Corchorus capsularis* and *C. olitorius*)

Chlorosis

Symptoms: Affected plants develop uneven surface and general yellowing with interspersed green areas. Severely affected plants remain stunted and either they do not flower or do not produce seeds. The disease is reported from India (Ghosh and Basak, 1951; Ghosh and Kundu, 1954; Bisht and Mathur, 1964).

Transmission: The virus is transmitted by grafting and by *Bemisia tabaci* (Bisht and Mathur, 1964). There is also indication of seed transmission of the virus (Ghosh and Kundu, 1954).

Yellow Mosaic of *Corchorus trilobularis*

Symptoms: Infected plants are slightly dwarfed and tend to flower early. Occasionally the flowers are sterile. In severe cases of infection the entire leaf becomes yellow (Varma *et al.*, 1966).

Transmission: In India the virus is transmitted by the whitefly, *Bemisia tabaci* (Varma *et al.*, 1966).

SUNNHEMP (*Crotolaria juncea*)

Mosaic

Symptoms: The virus (SMV) causes mottling, reduction in size and blistering of leaf. In severely affected leaves mesophyll is incompletely differentiated, chloroplasts remain indistinct and a

few phloem cells are hypertrophied. The affected plants become dwarf and produce few seeds (Raychaudhuri, 1947).

Transmission: The virus is sap transmissible.

Properties: It is inactivated between 68 and 70°C and loses infectivity *in vitro* after storage of 74–76 days and by dilution between 1: 1000–1: 5000 (Raychaudhuri, 1947). The virus particles are spherical with diameter ranging from 20 to 40 nm (Das Gupta *et al.*, 1951 and De, 1951). Das and Raychaudhuri (1963) studied the effect of UV on the infectivity of SMV. The virus infectivity has been also affected by mercuric chloride (upto 2 per cent), 90 per cent alcohol, chloroform, petroleum ether, 0.5 per cent copper sulphate (Das and Raychaudhuri, 1963) and NAA (Raychaudhuri and Mishra, 1963).

Southern Sunnhemp Mosaic (SSMV)

The virus, a strain of TMV (Capoor, 1962) was first described by Capoor (1950) from Poona, India.

Symptoms: It produces symptoms similar to those produced by SMV on *Crotalaria juncea*. Sometimes diseased leaves also develop enations and, the diseased plants yield less.

The virus differs considerably from SMV in other properties (Raychaudhuri *et al.*, 1962). SSMV has a wide host range (Capoor, 1950, 1962; Paliwal and Nariani, 1963). Guar is a local lesion host for both viruses (Raychaudhuri *et al.*, 1962).

Transmission: The virus is readily transmitted by mechanical inoculation and is also transmitted by root inoculation (Nariani and Chandrasekhar, 1963).

Properties: The virus is inactivated beyond 90°C and loses its infectivity at dilution beyond 10^{-6} and remains infective for eight years (Capoor, 1962). Treatment with 90 per cent ethanol for 24 hours did not impair infectivity (Capoor, 1950). Purified virus showed anisotropy of flow characteristic of liquid crystalline anisometric viruses (Capoor, 1962). Behaviour of this virus in tissue culture (Raychaudhuri and Mishra, 1965) has also been studied. SSMV is serologically related to bottlegourd mosaic, cowpea mosaic and tobacco mosaic viruses (Anand and Sahambi, 1965; Capoor, 1962). Although serologically related SSMV and TMV did not cross protect (Capoor, 1962). Electron micrographs of purified virus preparations revealed the virus particles measuring 300 nm \times 18 nm (Nariani *et al.*, 1970).

The growth of sunnhemp has been retarded at higher levels of phosphorus, virus concentration increased and greater number of local lesions have been produced in plants deficient in potash. Pre or post-inoculation darkening and carbon dioxide supply of guar plants also had effect on local lesion formation (Sastry, 1963). Effect of ultraviolet and gamma rays on virus infectivity has also been studied (Nariani and Paliwal, 1963).

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12

Forage Crops

BERSEEM (*Trifolium alexandrinum*)

Mosaic

Symptoms: In India the disease on berseem clover is characterised by typical mosaic and chlorotic streak appearance.

Transmission: The virus is sap transmissible with or without abrasive. The virus is also transmitted by *Aphis gossypii*.

Properties: The thermal inactivation point of the virus ranges between 45–54°C. The dilution end point of the virus is found to be 1:140 to 1:160. The virus particles are of bullet shape and measure 18–45 nm in length and 15 nm in diameter.

Host range: The disease is transmitted to petunia, brinjal, *Chenopodium amaranticolor*, *C. album*, tomato, *Nicotiana tabacum*, *N. rustica* and bean (*Phaseolus vulgaris*).

Distribution: The disease has been observed in India.

LUCERNE (*Medicago sativa*)

Mosaic

Symptoms: Symptoms on the leaves consist of bright yellow-white mosaic of the 'Calico' type. Affected plants are dwarfed and the leaves distinctly mottled and crinkled (Feldman and Gracia Olga, 1970).

Transmission: The virus is sap transmissible. The insect vector is the aphid, *Macrosiphum pisi*.

Properties: It has thermal inactivation point of 42°C (Milbrath, 1959). A strain of Lucerne mosaic virus causing yellowing, blotching and stunting has been identified by Diachun and Henson

(1957). It has been shown to be the same as mechanically transmissible yellow spot virus, another strain of alfalfa mosaic. This new strain of lucerne mosaic causes yellow systemic stipple followed by large yellow spots and slight mottling on the trifoliate leaves of bean (*Phaseolus vulgaris*), small necrotic lesions on Pinto and Refugee beans. Bright yellow spots and mottling are produced on the trifoliate leaves of top crop beans and small white rings on the inoculated tobacco leaves.

The relationship of the alfalfa yellow mosaic strain to lucerne mosaic virus was established by cross-protection and serological tests. The physical properties resemble those of the potato calico and potato tuber necrosis strains (Zentmeyer, 1962).

PHASEOLUS ATROPURPUREUS

Mosaic

Symptoms: The leaves of the diseased plants show mosaic mottling consisting of irregular dark and light green areas, occasionally the interveinal areas become yellowish. As the leaves grow older, they show slight malformation and the dark green areas turn into raised blisters. These leaves later exhibit reddish irregular necrotic spots which are smaller in size in the beginning but later coalesce to form bigger necrotic areas. Pod setting is comparatively less in disease vines which are stunted in growth (Moses and Nariani, 1969).

Transmission: The disease is transmitted by sap inoculation using carborundum powder as an abrasive. Two species of aphids, namely, *Aphis craccivora* and *Rhopalosiphum pseudobrassicae* successfully transmitted the virus. The virus is also transmitted through seeds of cv. Siratro upto 8.3 per cent.

Properties: The virus has a thermal inactivation point of 62 to 65°C, dilution end point of 1:10,000 to 1:50,000 and longevity *in vitro* for one to two days at room temperature (27.2° to 37.4°C) and four days at 4 to 7°C. The virus withstands desiccation for seven days and continuous freezing beyond ten days. Thirty minutes treatment with 1:100 hydrochloric acid, 1:200 formaldehyde and 75 per cent alcohol inactivates the virus.

Host range: The host range of the virus is confined to the family leguminosae. The virus induced systemic mosaic mottling on *Phaseolus vulgaris*, *P. aureus*, *P. mungo*, *P. lathyroides*, *P.*

calcaratus, *P. acutifolius*, *Crotalaria spectabilis*, *Cicer arietinum*, *Vicia faba*, *Vigna sinensis*, *Melilotus parviflora* but is carried symptomlessly in *Phaseolus acutifolius*, *P. angularis*, *Clitoria ternatea* and *Vigna catjang* (Moses and Nariani, 1969).

• The causal virus is identified as a strain of common bean mosaic virus and reported from India.

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13

Forest Trees

POPLAR (*Populus* sp.)

Mosaic

Symptoms: The disease manifests as fine interveinal chlorotic spots and further develops to either mosaic or interveinal chlorosis depending on the clone (Biddle and Tinsley, 1968).

Transmission: Schmelzer (1966) showed that a wide range of herbaceous host plants are susceptible to infection with poplar mosaic virus by mechanical transmission.

Properties: The thermal inactivation point is above 79°C and the longevity *in vitro* is two days at room temperature (Schmelzer, 1966).

The virus particles are flexuous rods having a length of 679nm. The particles are liable to aggregate during purification; however, the virus can be purified by precipitation which is followed by density gradient centrifugation in sucrose or by gel filtration.

SANDAL (*Santalum album*)

Leaf Curl Mosaic

The disease has been observed in Bangalore and Mysore cities in India.

Symptoms: In field two stages of the disease are observed. In the first stage conspicuous mosaic spots develop between the veins of the leaves, which show slight rolling. Matured leaves show mottling, but not young leaves. The tree remains in this stage during one growing season. In the second stage the new leaves show ruffling at the edges even when quite young, and develop

wrinkled and mottled appearance as they grow older. Dwarfing of the leaves and bearing twigs become conspicuous, and the new leaves that develop become progressively smaller in size, and finally get curled inwards, while those at the tip bend outwards. The leaves become thickened and brittle and fall off prematurely (Venkata Rao, 1933).

Transmission: The virus is transmitted by ring-grafts, but not by sap inoculation (Venkata Rao, 1933).

SPRUCE (*Picea excelsa*)

Spruce Virosis

Symptoms: Infected trees develop shortened chlorotic needles and defoliation is commonly met with.

Transmission: The virus is transmitted by *Sacciphantes abietis* and *Cinara piceae pilicornis*.

Properties: Rod shaped particles have been found in the exudate from the twigs of Sitka spruce. These particles are similar in morphology to those of the virus described, by Cech *et al.* (1961) which is unique in its characteristics.

Host range: The host range includes *Pinus* spp., *Abies* spp., *Larix decidua* in addition to *Picea abies* (Biddle and Tinsley, 1968).

The virus is probably identical with that described from Norway spruce in Czechoslovakia where it is transmitted by *Adelges abietis*.

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14

Fruits

ALMOND (*Prunus amygdalus*)

Bisht and Gupta (1962) recorded a mosaic disease of almond from Kumaon hills, India while yellow vein of almond (almond mosaic) has also been recorded on kagzi almond plants from Simla region by Nagaich and Vashisth (1965b).

Symptoms: The disease manifests as yellow flecking at the margin of leaves in the early season. Later the veins become yellow and characteristic yellow-net of veins develops on part or entire leaf lamina. Sometimes the leaf becomes yellow either partly or in full.

Transmission: The virus is not sap-transmissible but can be transmitted by bud grafts (Nagaich and Vashisth, 1965b).

APPLE (*Pyrus malus*)

Bunchy Top

Symptoms: The symptoms consist of complete suppression of internodes in the growing axis with the result that the leaves are clustered as a 'bunch'. The leaf size is much reduced. The diseased plants remain stunted and vigour is reduced considerably.

Transmission: Sharma from Simla observed that the disease is graft transmissible only. (Personal communication).

Leaf Pucker

Symptoms: Symptoms consist of light yellow spots and flecking near the veins. Subsequently spots become necrotic, leaves

puckered and deformed (Nagaich and Vashisth, 1965b). The disease has been observed in Simla.

Transmission: The virus is graft transmissible. The disease resembles leaf pucker of apple reported from Canada (Welsh and Keané, 1957).

Little Leaf

Symptoms: The disease is characterised by smalling of leaves and internodes. Leaves show mottling and deformed leaf buds on affected branches which open later than on healthy ones. Some diseased buds do not open at all and tend to die (Nagaich and Vashisth, 1965b).

Transmission: The virus is transmitted by wedge-grafting. The disease has been observed in India.

Mosaic

Symptoms: The disease is characterised by hairy, creamy white or yellow patches produced on lamina which are clear against green background. Occasionally spots enlarge and the leaf turns yellow. Yellowing develops along primary and secondary veins. In several cases, mottling is milder or in the form of yellow white rings. As season advances leaves become necrotic and wither out (Nagaich and Vashisth, 1963).

On the basis of symptomatology three strains of the virus have been recognised at Simla in India.

Severe strain: Affected plants show large creamy white or yellow areas on the leaves. The infected plants are stunted. Sometimes leaves show deformity at margins in advanced stages.

Vein banding strain: The virus causes permanent yellow-net pattern with small flecks between the veins. Necrosis between the veins may develop and defoliation is rare.

Mild strain: In this case the symptoms consist of relatively fewer and smaller flecks. Vein-banding and chlorotic areas are absent and defoliation does not occur.

Transmission: The virus is sap and graft transmissible. Besides apple, the disease is transmitted by grafting to plum, peach and loquat.

Control: An effective control measure for this disease is the use of certified virus-free budwood. Heat therapy has been found to be effective in controlling the disease (Nagaich and Vashisth,

1963). Exposure of seedlings to $36 \pm 2^{\circ}\text{C}$ in an incubator for twenty-five to twenty eight days inactivated the virus.

Star Crack

Symptoms: The characteristic symptoms consist of star-shaped cracks on the fruit skin. In certain varieties star cracks develop around the buds on one year old shoots and the tips of affected shoots are killed. The blossoming is delayed (Dhingra and Raychaudhuri, 1970). The disease has been observed in Simla, India.

Transmission: The virus is bud as well as graft transmissible.

Control: The disease can be controlled by the use of virus free budwood.

APRICOT (*Prunus bokhariensis*)

Necrosis Leaf Roll

Symptoms: The disease is characterised by the development of necrosis and rolling of the leaves.

This virus has been observed in Cape Province, South Africa (Wolfswinkel, 1966).

Yellow Mosaic

Symptoms: This is a new virus and causes yellowing of the leaves. It has been recorded from apricot orchards in Cape Province, South Africa (Wolfswinkel, 1966).

ARTICHOKE (*Cynara* sp.)

Mosaic

Symptoms: The disease is characterised by the presence of bright yellow circular, elliptical, polygonal or irregular spots, scattered over the entire leaf blade and ranging from 1 to 7 mm in diameter. Sometimes two to three spots become confluent (Gigante, 1951). This disease has been reported from Italy.

Transmission: The virus is transmissible through sap inoculations.

Host range: *C. scilymus* and *C. cordunculus* CV. *utilis* on infection gave rise to typical symptoms in three weeks.

BANANA (*Musa* sp.)**Mosaic**

Symptoms: The disease is characterised by the presence of chlorotic mottling, mosaic and large yellow streaks across the lamina on affected plants (Capoor and Varma, 1958, 1970). The disease is prevalent in India.

Transmission: The causal virus is not transmitted to banana by mechanical inoculation but is readily transmitted by *Aphis gossypii*. It is, however, transmitted mechanically to cucumber but the virus can not be recovered back on to banana. The virus is of non-persistent type.

Host range: Only banana cv. Lla vazahi, *M. salbisiana*, *M. chilcarpa*, *M. coccinia* and *M. acuminata* proved resistant (Capoor, 1967a, Capoor and Varma, 1958, 1970).

Control: The disease can be controlled by destroying infected suckers and by selection of disease free suckers for planting.

Bunchy Top

Symptoms: The infected plant shows bunching of the leaves at the apex to form a rosette. The rosette effect is due to failure of the leaf stalks to elongate. Initially the symptoms appear as irregular nodular green streaks along the secondary veins of the leaf sheath. The fruit bunches usually do not come out or may emerge with difficulty (Capoor, 1967b). The disease is very serious specially in South India, Australia, Ceylon, Fiji and Egypt.

Transmission: The virus is not sap transmissible but is transmitted by the banana aphid, *Pentalonia nigronervosa* (Capoor, 1967a).

Chemical analysis: Soil samples from the base of infected plants are more acidic and have a higher content of organic matter, nitrogen, phosphorus; but a lower content of total potassium and calcium with very little magnesium. Infected leaves have large amount of nitrogen, phosphorus and a very high content of potassium (Nambiar *et al.*, 1965).

Host range: Almost all the cultivated varieties of banana including some wild varieties are susceptible.

Control: Destruction of diseased plants and use of virus free suckers for new plantings are desirable.

CAPE-GOOSE BERRY (*Physalis peruviana*)**Mosaic**

Symptoms: Symptoms on *Physalis peruviana* include dwarfing of plant, puckering and blistering of leaves and premature shedding of leaves and fruits (Capoor and Sharma, 1965).

Transmission: The virus is sap transmissible.

The cape-goose berry mosaic virus (CGMV) is not transmitted by *Myzus persicae*, *Aphis rumicis*, *A. gossypii*, *Macrosiphum pisi* and by an unidentified grass hopper. It is neither transmitted through soil nor is seed-borne but is transmitted to a very high percentage through plucking of leaves from infected and healthy plants simultaneously. The virus has been shown to be a strain of TMV and it occurs in India.

Properties: The virus shows remarkable resistance to heat, i.e., it is completely inactivated by exposure to heat at 96°C. The virus withstands dilution upto 1: 10⁶ but is rendered inactive when diluted upto 1: 10⁷. The longevity *in vitro* of the virus diluted 1: 1 with water is 2 years at room temperature (20-32°C).

In addition a mosaic disease caused by cucumber mosaic virus has been reported (Singh, 1968; Nariani and Sharma, 1971).

Leaf Curl

Symptoms: The disease is identified by curling, puckering and smalling of the leaves and, presence of dark green enations on the veins underneath. The veins and veinlets are very prominent and thickened. The enations are oval or cup shaped, frilled and sessile. In severe cases the margins of the leaves are curled upwards forming cup like structures but in mild cases curling is only slight however, the enations are always present.

Transmission: The disease is graft transmissible and is not transmitted through juice extracted from the leaves of diseased plants. The whitefly (*Bemisia tabaci*) acts as the vector.

Host range: The virus is transmitted to *Nicotiana tabacum* cv. Harrison's Special by grafting as well as by the agency of whitefly (Nariani and Pathanian, 1953). The disease occurs commonly in India.

CHERRY (*Prunus* sp.)**Amasya Cherry Disease**

Symptoms: The affected trees become stunted and show die-back symptoms. In case of severe disease development affected trees die within three to five years. Yellowish green or oily spots 1–2 cm in diameter develop on mature leaves which may coalesce. The spots on the affected leaves are rusty orange to brick red coloured in contrast with the dark green of the rest of the leaf. Heavy defoliation may occur near harvest time and necrotic spots develop in the inner bark (Blodgett *et al.*, 1970). This disease has been reported from Turkey.

Transmission: The disease is transmitted by graft inoculation.

Little Cherry

Symptoms: Symptoms of the disease consist of early autumn leaf colouration and small unevenly ripening fruit with lack of flavour. Virus indicators are varieties Sam, Van and 12/1 of *Prunus avium* (Posnette, 1964). The disease has been reported from British Columbia and may be due to mycoplasma.

Transmission: Three species of leaf hopper, especially *Macrostelus fascifrons* are the vectors of the virus (Wilde, 1960).

Tatter Leaf

Symptoms: Symptoms consist of chlorotic ring spots on the leaves which later become necrotic. After sometime the necrotic tissues fall off leaving holes in the leaves (Nagaich and Vashisth, 1965b). The disease has been reported from Simla.

Transmission: The virus is transmitted mechanically to the Squash cv. Butternut.

CITRUS (*Citrus* spp.)**Crinkly Leaf**

Symptoms: Lemon shows warping and pocketing of the mature leaves due to irregularities of growth in different parts of the leaf blade. The intensity and distribution of symptoms vary in the affected leaves. Some trees show symptoms on one or two branches only and rest of the branches bear healthy leaves. Severely affected trees have upright, restricted growth.

Nearly all kinds of citrus are hosts but some show slight or no symptoms. Eureka and Lisbon lemon plants develop small circular pin-point spots on young leaves followed by crinkling. Pin-point spotting is persistent but not present on all leaves. Lemon fruits are sometimes small and misshapened (Fraser, 1961).

Transmission: The disease is graft transmissible. Mechanical transmission has been successful to various citrus species and to *Vigna sinensis* var. Black eye. The latter reacts with formation of local lesions (Daniello Daughy and Bove, 1965).

Recently the disease has been reported from India (Ahlawat and Sardar, in press).

Leaf Mottle or Leaf Mottle Yellows

Symptoms: The disease causes leaf mottling, yellowing, twig die-back and death of affected trees (Martinez and Wallace, 1967). The disease has been reported from Philippines. This is caused by mycoplasma-like organism (MLO).

Transmission: The causal agent is transmitted by grafting (Salibe and Cortez, 1967) and by the citrus psyllid *Diaphorina citri* (Martinez and Wallace, 1967).

Exocortis (Viroid)

Exocortis of citrus was formerly known to be due to virus. However, recently it has been established that this malady is due to a new entity called viroid.

Viroids are replicating free infectious RNA molecules of low molecular weight, which may causes pathological conditions in their hosts. They are characterised by the apparent absence of a dormant phase (i.e., virion) and by genomes that are much smaller than those of known viruses (Diener, 1971). Sedimentation properties and nuclease sensitivity of the infectious material and the absence of virions provide the basic evidence for the presence of viroids. The agent of potato spindle tuber disease (PSTV) was the first viroid discovered (Diener and Reymer, 1967). Later on the agents of citrus exocortis disease (Semancik and Weathers, 1968) and chrysanthemum stunt disease (Lawson, 1968) have been reported to belong to this category.

The evidence presented by Semancik and Weathers (1972b) suggest a hair pin-like structure of a partial double stranded structure with single stranded out pockets, e.g., CEV.

Symptoms: Affected trees show cracking and scaling of bark on susceptible root stocks which extend to the ground level. The plants are stunted (Nariani *et al.*, 1968). The branches of infected Rangpur lime show yellowish elongated blotches followed by splitting of the bark with curled edges. Bark splitting also develops on infected sweet lime. The disease has been reported from India.

Transmission: The dissemination of the pathogen is principally through infected budwood. It exists as a free ribonucleic acid (RNA) without a protein sheath (Semancik and Weathers, 1968, 1970). No insect vector is known. The viroid has been found to be carried on pruning and budding knives and also transmitted by dodder *Cuscuta subinclusa* from citrus to Petunia.

Properties: The causal agents of citrus exocortis disease, i.e., viroid are characterised by long incubation periods, by a relatively high heat resistance and by their association with nuclei, particularly chromatin of infected cells. No conventional viruses are found in exocortis infected plants and the agent can only be extracted efficiently with phenol or highly alkaline salt solution. Their infectivity is susceptible to inactivation by RNase but not by DNase. They can be readily concentrated by ethanol precipitation followed by resuspension. They are insensitive to treatment with various organic solvents like phenol, etc. These properties together with their low sedimentation rates in sucrose gradients and their behaviour in column chromatography suggested that they are free infectious RNA molecules of low molecular weight.

The exocortis RNA appears to exist in a non-disperse state representing molecules with molecular weight of 50,000 to 60,000 daltons, with chain length of 160–200 nucleotides (Sanger, 1972). However, Semancik and Weathers (1972a) reported the molecular weight of exocortis RNA as 125,000 daltons as estimated from migration in 5 per cent gels.

Infectious Variegation

Symptoms: The disease is characterised by leaf crinkling, distortion and blotching. The leaves often show pin point spots as in crinkly leaf. All plants showing infectious variegation symptoms appear to contain crinkly leaf which can be separated from the complex. The disease has been reported from Florida.

Transmission: By mechanical inoculation as well as by grafting.

Host range: The virus infects several citrus species. The disease

has been mechanically transmitted to Eureka lemon, sour orange, grape fruit, key lime, *Citrus grandis*, *Crotalaria spectabilis* and *Vigna sinensis* (Grant and Corbett, 1965). It has been demonstrated that crinkly leaf and infectious variegation are closely related.

Leaf Curl

The disease has been reported from Brazil (Salibe, 1959).

Symptoms: The striking symptoms of the disease are curling and distortion of the leaves. Affected plants produce weak sprouts. At the origin of sprouts there is a deposition of a gum in wood vessels. Channelling and pitting of the wood of the trunk and main branches also appear in diseased plants. Finally infected branches die-back. The new leaves are small and curled. The diseased trees produce abundant flowers but the fruits are few and small. Indexing can be done on two to five months old sweet orange seedlings.

Transmission: The disease is transmitted by budding and grafting.

Host range: The virus is transmitted to sweet orange varieties, Tangerines, Eureka lemon, sweet lime, acid lime, sour orange, citron, grape fruit and shaddock (Salibe, 1965).

Control: The disease is controlled by uprooting and destroying infected trees.

Psorosis

The psorosis virus group consists of psorosis A, concave gum and blind pocket as they have certain common young leaf symptoms. Crinkly leaf and infectious variegation have been considered to be strains of psorosis virus (Wallace, 1957). Frazer (1961) concluded that crinkly leaf did not belong to the psorosis group but Wallace (1945) found that some kind of citrus develop characteristic young leaf symptoms of psorosis after inoculation with purified crinkly leaf virus and concluded that crinkly leaf can be grouped with the psorosis viruses.

Psorosis A

Symptoms: The virus induces vein flecking and vein banding symptoms on young leaves of sweet orange. The flecks may sometimes coalesce to form speck or blotchy patterns. The bark symptoms on sweet orange, mandarin and grape fruit trees are scales on outer bark layers or aggregates of small pustules under

which the tissue is brown. As the scaling advances, deeper layers of bark are affected by irregular growth and gum exudation. Gum deposits are formed within and between layers of wood, more or less corresponding to the seasonal and annular rings of growth and the wood under old lesions becomes darkly stained from gum impregnation.

Transmission The disease is transmissible by bud grafting as also by other methods of grafting such as root grafting, patch grafting and leaf tissue grafting (Wallace, 1945). No insect vector is known and the virus is not sap inoculable. It has been transmitted by dodder, *Cuscuta subinclusa* and *C. compacta* (Weathers and Harjung, 1964; Price, 1965).

Host range: The virus is confined to Rutaceae and infects all *Citrus* species. Lemon (*C. limon*) and sour orange (*C. aurantium*) develop leaf symptoms but no bark lesions.

Concave Gum

The disease is characterised by concavities of various sizes which develop on the trunks and larger limbs and the bark tends to crack round the rim of the cavity with gum oozing to the surface. The symptoms produced in young leaves are similar to those caused by psorosis A except that in spring growth most of the leaves show more of oak leaf pattern.

Blind Pocket

The bark symptoms usually appear as depressions. These sometimes run together forming furrows and the two opposite sides may grow together leaving a narrow depression. Young leaf symptoms are similar to those of psorosis A except that there are no bark lesions unless a tree is infected with a mixture of blind pocket and psorosis A viruses.

The disease is of almost world wide distribution.

Tristeza

Symptoms: The symptoms of the disease vary depending upon the *Citrus* species, the root-stock used and the strain of the virus present. The first symptom of the disease on susceptible root-stock-scion combinations such as sweet orange on sour orange is the partial or complete suppression of new flushes and premature coloration of fruits. The leaves become dull or slightly bronzed at

first, followed by yellowing, especially veinal chlorosis. Progressive defoliation follows towards the tips until many twigs are bare. Limbs begin to die back from the tips and weak shoots emerge from the axillary buds. The diseased trees have the tendency to blossom heavily in the earlier stage of infection. The affected trees also show severe root injury. The rootlets decay first followed by extensive rotting of larger roots. Some trees may show symptoms of sudden decline and death, thereby justifying the name Quick Decline. These symptoms result from necrosis of the sieve tubes immediately below the bud union.

Indicator hosts: Mexican (West Indian or Key) lime (*C. aurantifolia*) is the commonly used indicator host. In India it has been demonstrated that Kagzi lime is an ideal indicator plant for the virus (Capoor, 1961). The diagnostic symptoms consist of sharp clearing or flecking of veins and veinlets. Stem pitting of the twigs is seen when bark is removed.

Transmission: The virus is not sap inoculable but is transmitted by grafting and the aphid vectors such as *Toxoptera citricidus* and *Aphis gossypii* (Vasudeva *et al.*, 1959; Varma *et al.*, 1960 and 1965). The virus was transmitted by dodder *Cuscuta subinclusa* (Weathers and Harjung, 1964) and *C. reflexa* (Nariani and Raychaudhuri, 1970 and Sharma, 1971).

Properties: The virus particles measure $2000 \cdot 10-12$ nm as seen in electron micrographs. The sedimentation constant of 20 per cent of purified particles was 105-131 S but other procedures caused fragmentation of almost all particles (Kitajima *et al.*, 1965; Bar Joseph *et al.*, 1970).

Host range: Sweet orange, grape fruit, tangerine, mandarins, some tangelos on sour orange root stock and sweet orange on grape fruit rootstock are particularly susceptible. Rootstock studies in California and Brazil have shown that trees of sweet orange on many other species and varieties are susceptible.

Control: The only way to control the virus is to use resistant or tolerant root stocks and certified virus budwood for propagation. In India, in most regions, oranges, grapefruit, mandarins, lime and lemons are propagated either on strains of rough lemon (*Citrus jambhiri*) or Karna khatta (*Citrus karna*) which are comparatively tolerant to the virus.

Seedling Yellows

It is a virus disease found only in association with tristeza and may be caused by one or more forms of tristeza virus (Wallace, 1959). The disease occurs in Australia, California and South Africa and, has also been recorded from India; and is suggested to be caused by a strain of tristeza virus (Capoor, 1965).

Symptoms: It is characterised by the appearance of stunting and yellowing of seedlings. Leaves of severely affected plants appear small, greenish yellow and remain underdeveloped. Internodes of the stem and the shoots become short and multiple buds may develop at the nodes (McClellan, 1960; Wallace, 1957, 1959; Capoor, 1965).

Transmission: The virus is transmitted to seedlings by grafting and by *Toxoptera citricidus*.

Host range: The virus infects sour orange, grapefruit, Eureka lemon, Citrus, sweet orange, South African lemon, Satsuma and some other mandarins, Waialua orange and Kumquat (Wallace, 1957) and it is widespread in Japan (Yamada and Tanaka, 1969).

Vein Enation

It has been shown that vein enation and woody gall are caused by the same virus although the two diseases were earlier reported to be different. The diseases are reported from California and South Africa.

Symptoms: Small swellings and distinct projections called 'enations' appear on the under surface of the leaf veins. The upper surface shows corresponding depressions. The number and size of the enations varies depending upon the variety of citrus affected and possibly strain of virus.

Most of the commercially used citrus scion and root-stock varieties are hosts of the virus although some develop no enations. Mexican lime (*C. aurantifolia*), some other sour lime varieties and sour orange (*C. aurantium*) develop pronounced enations. Rough lemon and sweet orange develop slight enations in the green house but show less conspicuous symptoms in the field.

Woody Gall

Galls appear on twigs as slight swellings over which the bark becomes greyish. On Mexican lime and rough lemon, galls develop in the main stem usually close to the thorns. As they

increase in size, these become irregular in shape. The bark becomes rough and light coloured. Adjacent galls may coalesce to form cauliflower like structures.

The galls appear commonly on Rough lemon and Mexican lime.

Transmission: The virus is transmitted by three species of aphids, *Toxotera citricidus*, *Myzus persicae* and *Aphis gossypii* and by grafting.

Xyloporosis (Cachexia)

The disease occurs in Palestine, Brazil and Florida.

Symptoms: On removing the bark of susceptible root stocks, peg like projections are noticed in the inner face of bark with corresponding pits within the xylem. Gum impregnation of the phloem tissues accompanies these symptoms which may cause partial girdling. The secondary symptoms consist of yellowing of the leaves, early blooming and fruiting and partial leaf drop due to girdling.

Transmission. The virus is transmitted by grafting only.

Indicator plants: Rangpur lime, Palestine sweet lime and Orlando tangelo are good indicator plants.

Control: Avoid Rangpur lime and sweet lime root stocks. Use virus-free buds for propagation.

FIG (*Ficus* sp.)

Mosaic

Symptoms: The disease is characterised by the development of yellowish green spots scattered all over the leaf lamina. These coalesce to form bigger spots of various shapes and sizes. Occasionally the leaves develop white mottle and are deformed. The leaves of wild varieties develop marked mosaic symptoms and sometimes have oak leaf pattern. The diseased plants produced very few fruits (Bhargava and Bisht, 1961; Nagaich and Vashisth, 1962).

Transmission: The virus is graft transmissible (Flock and Wallace, 1956). The mite *Acaria ficus* was found to transmit fig mosaic to *F. carica* (Vashisth and Nagaich, 1968).

Host range: The virus has been transmitted to *Ficus palmali*,

F. carica, *F. nemoralis* and *Morus indica* (Vashisth and Nagaich, 1965).

Mulberry developed mosaic mottling by graft inoculation, with fig mosaic virus (Vashisth and Nagaich, 1965). The virus has recently been reported to occur in India.

GRAPEVINE (*Vitis vinifera*)

Pierce' Disease (*Anaheim disease*)

The disease was first recorded from Anaheim in 1884-85. Until 1953 the disease was restricted to California only. Later it was recognised in Florida and Argentina.

Symptoms: The symptoms consist of drying up of leaves from the margin to the petiole and complete browning of leaf lamina. Premature colouring of berries, delayed foliation in the spring, dwarfing of entire or part of the vine and gradual dying of the root system followed by wilting of affected plants are also observed.

Transmission: The virus is neither sap transmissible nor seed-borne. It has been successfully transmitted through grafting. Four sharp shooter leafhoppers, namely *Hordnia circellata*, *Draculacephala minerva*, *Carniocephala fulgida* and *C. flaviceps* are the important vectors of the virus in nature. Other leafhoppers reported to be vectors are *C. triguttata*, *Cucerna occidentalis*, *Graphocephala cythura*, *Holochara delta*, *Homalodisca liturata*, *Neokolla gothica*, *N. confluens*, *N. heiroglyphics*, *Pagaronia trunata*, *P. confusa*, *C. yuccae*, *D. californica*, *D. navchorae* s and *D. orassicernis*.

Host range: The virus has wide host range but most of the hosts are symptomless carriers. Alfalfa and snowberry (*Symphoricarpus albus*) are useful as they develop definite symptoms. Alfalfa suffers a great loss due to alfalfa dwarf disease incited by the virus.

Control

1. Cuttings for propagation should be obtained from certified vineyards.
2. The vineyards should be free from weeds or alfalfa which serves as reservoir for the virus.
3. The vineyards should be sprayed with insecticides to keep away the vectors.

Mosaic (White mosaic)

The disease reported from California is the same as panachure (white mosaic) in France (Hewitt, 1945).

Symptoms: The diseased vines develop bright yellow leaves and these become chlorotic as they age. Diseased vines of certain varieties fail to set fruits.

Transmission: The virus is graft transmissible and no insect vector has been recorded. The disease spreads very slow in vineyards.

Fan leaf

The disease was observed on the variety pinot chardonnay from California in 1948.

Symptoms: The gradual dwarfing of the vines is the most characteristic symptom of the disease. The leaves and shoots get deformed in various ways or the setting of almost seedless berries may take place.

Transmission: The nematode, *Xiphinema index* transmits the disease (Hewitt *et al.*, 1958).

Control: Raski *et al.* (1971) advocated application of 1, 3-dichloropropane at 250 gal/acre in heavy soil for controlling the disease.

Asterioid mosaic

Symptoms: The disease incites mosaic symptoms on grapevine. The leaf spots are somewhat translucent and occur chiefly between the primary and secondary veins. The leaves are often malformed and margins are deep-cut, and in some varieties green blisters occur. During summer leaf symptoms become less severe but affected varieties are often stunted and fruit setting is poor (Hewitt and Goheen, 1959).

Transmission: Asterioid mosaic virus is transmitted by chip budding or grafting and has been reported from California

MULBERRY (*Morus* sp)**Mosaic**

Symptoms: The typical mosaic symptoms are frequently accompanied by slight curling and puckering of the leaves without any appreciable reduction in the leaf size (Raychaudhuri *et al.*, 1962).

Transmission: The disease is mechanically transmissible (Raychaudhuri *et al.*, 1965). In addition the aphids, namely, *Rhopalosiphum maidis*, *Myzus persicae* and *Aphis gossypii* have been shown to be aphid vectors of mulberry mosaic virus (Chatterjee and Raychaudhuri, 1963; 1965).

An unidentified aphid species obtained from *Hibiscus rosasinensis* and *Chrysanthemum* sp. is responsible for the transmission of the disease. *R. maidis* is capable of transmitting the virus without pre-acquisition fasting while *M. persicae* requires pre-acquisition fasting, which indicates *M. persicae* is a less efficient vector (Chatterjee and Raychaudhuri, 1965).

Properties: The virus can tolerate exposure upto 56°C for ten minutes but becomes innocuous when exposed to 50°C for the same period. It withstands dilution upto 1:2000, but is rendered inactive when diluted to 1:4000. The longevity *in vitro* of the virus is ten to fifteen days at room temperature ranging from 11 to 28°C (Raychaudhuri *et al.*, 1965).

Host range: The virus could be transmitted mechanically to 17 varieties of *Morus*, namely, *M. alba*, *M. bombycis*, *M. latifolia*, *M. macroura*, *M. mongolica*, *M. nigra*, *M. laevigata* and *M. multicaykes*. Mulberry varieties Ichinose and Kairynezumegaishi of *M. alba* and Oshimasho and Kosen of *M. latifolia* are, however, resistant to the virus (Raychaudhuri, *et al.*, 1965). This disease is quite common in India.

Yellow Net Vein

Symptoms: The disease is characterised by typical yellow net vein symptoms, yellowing of veins and veinlets, associated with chlorotic areas starting from the margins of affected leaves.

Transmission: The virus is not sap transmissible. The virus is easily transmitted from *M. indica* to *M. indica* by inarch grafting and by white fly *Bemisia* sp. (Raychaudhuri *et al.*, 1961). The disease has been found to occur in India.

Control: Heat and chemotherapy and resistant varieties may prove to be effective for controlling the disease.

PAPAYA (*Carica papaya*)

Symptoms: Affected plants are stunted with reduction in leaf number and size. Young leaves occasionally show vein clearing.

In severe cases the youngest leaves are distorted, crinkled, and blistered. Fruits often wither and abscise.

Decline: The disease has been reported from Kenya and East Africa (Kulkarni, 1970).

Transmission: The virus is transmissible by sap inoculation and grafting. Three viruses were isolated and differentiated on type of local lesions produced on *Chenopodium quinoa*. These are designated as chlorotic lesion virus (CLV), chlorotic ringspot virus (CRV) and necrotic lesion virus (NLV). Insect transmission tests with different insect vectors have negative results.

Properties: Thermal inactivation point of CLV, CRV and NLV are 60–65°C, 55–60°C and 75–80°C respectively. The maximum infective dilution for the three viruses are 1: 2000, 1: 22000 and 1: 22000 and survival *in vitro* eight days, six days and one day respectively at 14–22°C. The virus particles of all the three viruses measure 750×12 nm and are stiff rods.

Host range: The viruses could infect soybean, *Arachis hypogaea*, *Chenopodium quinoa* and *C. amaranticolor*.

Leaf Curl

Nariani (1956) made a detailed study of a leaf curl disease earlier reported by Thomas and Krishnaswamy (1939) from India and found the causal virus to be the same which causes leaf curl of tobacco.

Symptoms: Affected plants show severe curling, crinkling distortion of leaves, leaf margins rolled downwards and inwards and vein thickening. The leaves become leathery, brittle and the petioles are twisted in a zigzag manner. The severely affected plants fail to flower or bear very few fruits, in advanced stages of disease defoliation takes place and the growth is stunted.

Transmission: The disease is not sap transmissible but readily transmitted by grafting and *Bemisia tabaci*. The virus is experimentally transmitted to tomato and tobacco. The virus has a very wide host range and infects sunnhemp, chilli, *Petunia*, *Zinnia*, *Datura stramonium* and several other weeds and ornamental plants.

Control: Capoor (1967b) suggested possible control of the disease by cutting down the diseased plants in early stages of the disease development. No other method of control is known at present.

Mosaic

There appears to be a good deal of confusion regarding mosaic and ring spot diseases of papaya. Mosaic disease reported from Puerto Rico (Adsuar, 1946a, b), mosaic disease reported from India (Capoor and Varma, 1948), mosaic disease in Venezuela (Pontis Videla, 1953), Waiaina disease and ring spot reported from Hawaii (Parris, 1938; Jensen 1949a, b) and distortion ring spot and faint mottle ring spot from Florida and India (Conover, 1962, 1964, Khurana and Bhargava, 1970b) all these seem to be caused by one virus or strains of the same virus as they resemble closely in symptomatology, vector transmission, host range, physical properties and particle morphology.

Symptoms: The disease is characterised by mosaic symptoms on the leaves with blister like patches of green tissue distributed all over the yellowish green lamina. The younger leaves are very much reduced in size, become chlorotic and malformed often lamina reduced to a filiform shape. Conspicuous dark green spots and elongated streaks of water soaked or oil spots are formed on petioles and stem of diseased plants. Fruits develop circular or concentric rings or water soaked lesions with a central solid spot. The infected plants show degeneration and marked reduction in growth. The older leaves gradually fall off leaving the trees almost denuded except for a tuft of small leaves (Capoor and Varma, 1958). The distortion ring spot strain induces apocarp and double papaya fruit formation (Khurana and Bhargava, 1970b). Pinwheel shaped inclusions have been observed in ultra-thin sections (Zettler *et al.*, 1968).

Transmission: The disease is transmitted by mechanical sap inoculation as also by grafting and is aphid borne. Several species of aphids, namely, *Myzus persicae*, *Aphis gossypii*, *A. idicaginis*, *A. rumicis*, *A. mulvae*, *Macrosiphum solanifolii*, *M. sonchii*, *Micro-myzus formosanus* are known to be vectors (Jensen, 1949b; Capoor and Varma, 1958). The virus is not retained in *Myzus persicae* and *Aphis gossypii* for more than one to two hours. It is not soil borne.

Properties: The virus is inactivated by ten minute exposure to 53°C but not at 55°C. The dilution end point lies between 1: 1000 and 1: 10000 and the virus survives aging *in vitro* for twenty-six hours at room temperature but not twenty-eight hours.

Purified virus preparations revealed the particles to be long

flexuous rods of 800 nm (Herold and Wiebel, 1962; DeBokx, 1965). Khurana and Bhargava (1970b) report the particles of distortion ring spot in dip preparations to be 763 nm in length.

Host range: The virus has been transmitted to cucurbitaceous hosts (Capoor and Varma, 1958; Herold and Weibel, 1962; Conover, 1962). These include, *Cucumis sativus*, *C. melo*, *Lagenaria ciceraria*, *Cucurbita pepo*, *C. pepo* var. *melullosa*, *Citrullus vulgaris*, *C. fistulosus* and *Luffa acutangula*.

Control: *Carica cauliflora* has been found to be immune to infection with the virus (Capoor and Varma, 1961). If this immunity is gene controlled a breeding programme with cultivated varieties may help in evolving a resistant variety. Weekly sprays with one per cent groundnut oil help in preventing infection by aphids (Bhargava and Khurana, 1969).

Mild Mosaic

Symptoms: The disease is characterised by green mottle without leaf distortion. No symptoms on petiole, stem or fruits have been observed (Conover, 1962).

The virus has so far been recorded from Florida and India (Conover, 1962; Khurana and Bhargava, 1970a).

Transmission: The virus is transmitted by sap inoculation but not by *Myzus persicae*.

Properties: The virus is inactivated at 73–76°C., at a dilution beyond 1:20000 and is infectious after 187 days ageing (Conover, 1962). The particle size is 533 nm (DeBokx 1965).

Host range: Besides *Carica* spp, the virus infects *Meliohria pendula*, cucumber, musk melon, watermelon, *Antirrhinum majus* and *Sesamum indicum*. *Gomphrena globosa*, *Cassia occidentalis* and *Chenopodium amaranticolor* are local lesion hosts (DeBokx, 1965; Zettler *et al.*, 1968). *Vinca rosea* and zinnia are symptomless carriers (DeBokx, 1965).

PASSIFLORA FOETIDA

Yellow Vein Mosaic

Symptoms: The disease is characterised by clearing of veins and veinlets and has been reported from India (Wilson and Satyarajan, 1970).

Transmission: The virus is transmitted by grafting, but not by

sap inoculation. Further information is lacking.

PEACH (*Prunus persica*)

Mosaic

Symptoms: Affected plants show veinal chlorosis with yellow mottling. Mosaic affected leaves are crinkly. The trees are dwarfed and show profuse branching with short internodes.

Transmission: The virus is transmissible by grafting or budding but not by mechanical means. An eriophyid mite *Eriophyes insidiosus* (Wilson *et al.*, 1955) acts as a vector. Mites retain infectivity for two days and must feed on seeds rather than leaf blades to transmit the virus (Slykhuis, 1969). A peach mosaic virus has been reported from India to be graft transmissible (Bhargava and Bisht, 1960).

Host range: The virus infects almond, apricot, plum and nectarine.

PEAR (*Pyrus communis*)

Mosaic

Symptoms: The disease is identified by development of transient mottle, chlorotic spots and oak leaf pattern. Sometimes confluent rings are also observed (Bhargava and Bisht, 1961; Bisht and Gupta, 1962).

Transmission: The virus is transmitted by budding and grafting and has been reported from India.

PLUM (*Prunus domestica*)

Creamy White Spots

Symptoms: The virus is characterised by the presence of numerous creamy white spots. The spots are scattered all over the lamina (Nagaich and Vashisth, 1965a).

Transmission: The virus is not mechanically transmitted but is transmitted by budding to the varieties, Beauty and Santa Rosa. It has been reported from India.

Enation Mottle

Symptoms: Affected plants show mottle and chlorotic spots on lamina which later become necrotic. These are depressions on

the dorsal surface of the leaves with corresponding swellings on the ventral surface (Nagaich and Vashisth, 1965b).

Transmission: The disease is graft transmissible.

Host range: The disease is not transmitted by sap to any of the fruit trees and herbaceous plants tested. The disease has been reported from India.

Line Pattern

Symptoms: Virus is characterised by vein yellowing of leaves, sometimes mixed with oak leaf-pattern. In many cases chlorosis of the entire leaves accompanied by dwarfing of the plants has been encountered.

Often chlorosis was observed on green stems and petioles top. In many aerial rooting on the old branches is also noticed (Azad, 1960; Azad and Sehgal, 1959; Bhargava and Bisht, 1957a, b).

The disease has been reported from Simla and Kumaon hills, India.

Transmission: The virus is graft transmissible.

Host range: Plum, peach, cherry and *Prunus pudum* are infected by the virus.

Control: Heat therapy and use of virus free bud wood may be effective in controlling the disease.

Mosaic

Symptoms: These consist of light green mosaic, ringspot mottle, smalling and deformation of leaves.

According to Nagaich and Vashisth (1965b) it is a disease of complex nature incited by two different viruses, i.e., line-pattern and ring spot.

Transmission: The virus is transmitted by grafting.

Host range: The disease is transmitted to *P. persicae* and *P. pudum* as also to plum varieties green Cage, Beauty Smith and Green, and Satsuma plum. The transmission was also indicated in plum var. Beauty (Azad and Sehgal, 1958). The disease has been reported from India.

Leaf Roll

Symptoms: The disease is characterised by upward rolling of leaves and was observed in India (Mishra, 1966).

Transmission: The disease is transmitted by tongue grafting.

Host range: The virus infects plum, peach and apricot. The disease appears to be similar to the plum leaf roll reported from U.K. (Posnette, 1953).

Ring Spot and Shot-hole Virus

Symptoms: The disease is characterised by the appearance of yellow ring encircling green areas, which later turn brown and necrotic. These necrotic spots then drop leaving 'shot holes' on the leaves.

Transmission: The disease could not be transmitted by repeated mechanical inoculations. The virus is graft transmissible.

Host range: All the varieties of *Prunus domestica* tested were found to be susceptible. Symptoms appeared when such plants were inoculated with virus affected peach chips (Dhingra, 1972). This disease has been reported from India.

RASPBERRY (*Rubus ellipticus*)

Mosaic

Symptoms: A severe disease characterised by yellow or light green mosaic, ring spot mottle and oak-leaf pattern on the leaves has been reported from Simla hills, India (Azad and Sehgal, 1957).

Transmission: The disease has been successfully transmitted by inarch-grafting as well as by bottle-graft technique. It is also transmitted through dodder (*Cuscuta reflexa*) (Azad and Sehgal, 1957).

Chenopodium quinoa and *C. amaranticolor* developed local lesions after sap inoculation.

Ring Spot

Symptoms: The disease under field conditions consists of irregular chlorotic rings on leaves accompanied by the stunting of the affected plants. In addition, symptoms of mosaic, yellow net oak-leaf pattern and acute vein-clearing are also observed on a large number of plants.

Transmission: All attempts to transmit the virus by mechanical inoculation on herbaceous hosts as well as raspberry failed. The virus could not be transmitted by seed or root grafting. The disease, is, however, not transmitted either by aphids or whiteflies. The causal virus is transmitted through infected soil. Indication

of possible association of *Xiphinema* and *Longidorus* species suggest that a nematode may, perhaps, be acting as a vector (Dhingra and Niazi, 1972). The disease has been recently reported from India.

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15

Medicinal Plants

MADAR (*Calotropis gigantea*)

Mosaic

Symptoms: The disease is characterised by scattered, yellow, irregular spots appearing on the leaves, gradually enlarging and often coalescing to form large, irregular, yellow patches (Wilson and Jose, 1967).

Transmission: The virus is sap transmissible and is also transmitted by grafting (Wilson and Jose, 1967). No further information is available.

COFFEE SENNA (*Cassia occidentalis*)

Ring Spot Mosaic

Symptoms: The naturally infected plants are stunted and sickly, with light and dark green mosaic patterns on young pinnate leaves. The chlorotic patches become sharper and interspersed with ringspots on older leaves which are smaller and leathery as compared to healthy leaves.

Transmission: The virus is transmitted through sap only.

Properties: The virus remains infective on heating for ten minutes at 70°C but not at 80°C, on storage for eighteen hours *in vitro* (28–35°C) and four to six days at 9–10°C. It tolerates a dilution of 1:10⁻⁵ but not 1:10⁻⁶. The virus is not viable in desiccated leaves after four days. It resists alcohol and does not become innocuous when kept in 95 per cent alcohol for three hours.

Host range: The virus infects only a few hosts of economic importance belonging to the family leguminosae and is

characterised by local and systemic ring spots on cowpea T21 occasional rings and mosaic on *Cassia tora* but a simple mosaic pattern in other (Mathur and Singh, 1972).

The disease is reported from India.

THORN APPLE (*Datura metel*)

Enation Mosaic

Symptoms: The disease is characterised by puckering, distortion and enations on the leaves, abnormal flowers and suppression of the spines on the fruits (Verma and Verma, 1963). *Datura metel* mosaic disease from Mysore has also been described (Yaraguntiah and Govindu, 1972).

Transmission: The virus is transmitted by sap inoculation as well as by *Myzus persicae* and *Aphis gossypii* and is of the non-persistent type.

Properties: The virus is inactivated by dilution of 1:10,000. Thermal inactivation point is in between 60 to 70°C and longevity *in vitro* of five days at room temperature (23–30°C).

Host range: *Nicotiana glutinosa*, *N. tabacum*, cv. White Burley *N. plumbaginifolia*, *Petunia* sp. and *Solanum nigrum* are also found to be susceptible to it.

The virus is regarded as a strain of Datura virus 3.

DATURA ALBA

Distortion Mosaic

Symptoms: The infected plants become yellow in appearance due to the leaves which become yellowish green at advanced stages of infection. Flowers are also severely malformed and distorted but the diseased plants are seldom dwarfed.

Transmission: The virus is readily transmissible by mechanical means. The virus is not seed transmissible. The aphid, *Myzus persicae* is the vector.

Properties: The thermal inactivation point is 60°C. The dilution end point is 1:10,000 and the longevity *in vitro* is thirteen days at 80°F. It withstands treatment with 95 per cent ethyl alcohol for thirty hours at 45°F.

Host range: Tobacco, petunia, potato, *Datura alba*, *D. stramonium*, *D. fastuosa* and *N. glutinosa* are found to be

the hosts (Capoor and Varma, 1948).
The disease has been reported from India.

RAJWOLFIA SERPENTINA

Bunchy Top

The disease in India is characterised by newly formed leaves turning chlorotic, but the lower mature ones remain unaffected. Normally dormant axillary buds produce thin chlorotic bunches with usually long internodes. The main stem is stunted and the inflorescence is malformed or transformed into leafy shoots (Varadarajan, 1967).
Transmission: The virus is transmitted by grafting and by insect. The vectors have not been established though the moths and hoppers are the main visitors of *R. serpentina*.

Host range: The virus infects *Vinca rosea*, *Nicotiana tabacum*, *Solanum melongena* and *Capsicum annum*. The symptoms of the disease resemble those caused by aster yellows virus (Varadarajan, 1967).

The disease has been reported from India.

SIDA CARPINIFOLIA

Infectious Chlorosis

Symptoms: The disease is characterised by the development of yellow blotches on leaves.

Transmission: The disease is not sap transmissible but can be readily transmitted by grafting and whitefly, *Bemisia tabaci*.

The virus is considered closely related to chlorosis of malvaceae studied by Orlando and Silber-Schmidt (1946) in Brazil.

The disease is reported from Brazil, Puerto Rico.

SOLANUM KHASIANUM

Mosaic

Symptoms: Infected plants are completely stunted and growth is retarded, showing the crowding of the branches and giving a bushy appearance. Severely infected plants either do not flower normally or flower late. Flowers in such plants are very few and are very much reduced in size which ultimately give rise to smaller fruits of irregular shape.

Transmission: The virus is sap transmissible and is successfully transmitted by three species of aphids, namely, *Myzus persicae*, *Aphis gossypii* and *A. evonymi*.

Properties: The virus is inactivated when exposed to 60°C for ten minutes. The virus can withstand dilution upto 1:1000 but loses its infectivity at 1:10,000. The virus remained infective beyond ten days in the infected leaves stored at room temperature in a desiccator.

Host range: The virus infects the following hosts, namely, *Nicotiana tabacum* cv, White Burley, *N. glutinosa*, *Solanum aviculare*, *S. laciniatum*, *S. xanthocarpum*, *S. nigrum*, *Datura metel* var. *tatula*, *D. stramonium* var. *Godrii* (Thakur and Sastry, 1971). The disease is reported from India.

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16

Millet

RAGI (*Eleusine coracana*)

Ragi Mosaic Virus 1

Symptoms: The virus causes stunting, severe mosaic and chlorosis. Affected plants become pale yellow and in severe cases brownish white.

Transmission: The virus is mechanically transmitted and is also transmitted by aphids (*Rhopalosiphum maidis*, *R. rufiabdominalis*, *Aphis gossypii*, *Myzus persicae* and *Macrosiphum granarium*).

Properties: The virus has a thermal inactivation point between 50 to 55°C. The dilution end point ranges between 1: 500 to 1: 750 and the longevity *in vitro* ten to twelve hours at room temperature (30–36°C). Virus particles are found to be flexuous rods measuring 667 ± 8 nm in length and 12–14 nm in diameter. The virus is serologically related to sugarcane mosaic and maize mosaic viruses (Subbayya and Raychaudhuri, 1970).

Host range: The virus infects *Pennisetum typhoides*, *Setaria italica*, *Sorghum vulgare*, *Zea mays* and *Paspalum scrobiculatum*. The disease has been reported from Mysore, India.

Ragi Mosaic Virus II

Symptoms: Diseased plants develop typical mosaic symptoms. The diseased plants are stunted, the leaf size reduced and margins often curled inwards.

Transmission: The virus is readily transmitted mechanically but the vector is not known.

Properties: It has a thermal inactivation point between 45 to 50°C, dilution end point between 1: 50 and resistance to ageing of

six hours at room temperature (33–38°C).

Host range: The following plant species are found to be susceptible to the virus: *E. coracana*, *Sataria italica*, *Zea mays*, *Saccharum officinarum* and *Panicum miliaceum* (Batra *et al.*, 1966).

The disease occurs in India.

Eleusine Mosaic Virus

Symptoms: Virus induces mosaic symptoms in leaves. Diseased plants are stunted and ear heads of severely affected plants are malformed. Such plants produce few seeds of smaller size.

Transmission: The virus is readily transmitted mechanically and by the aphids, *Rhopalosiphum maidis*, *Schizaphis cyperi*, *Toxoptera graminum*, *Tetraneura nigrabdominalis*, *Aphis gossypii* and *Myzus persicae*. The virus is non-persistent in *R. maidis*. Three aphid species, *Longiunguis sacchari*, *Pentalonia nigronervosa* and *Aphis fabae* did not transmit the virus. Virus is not seed-borne in nature.

Properties: The virus is inactivated by heating to 55 to 60°C, dilution to 1:15 and ageing for two hours thirty minutes at room temperature.

Host range: Following hosts have been experimentally infected with this virus: *E. coracana* var. H.I., *Sorghum vulgare* var. M. 473, *Zea mays*, *Euchlaena mexicana*, *Setaria italica* vars. H-1, H-2, H.K. 289, 282, *Setaria tomentosa*, *S. viridis*, *S. verticillata*, *Oplismenus burmanni*, *Urochloa marathensis* var. *Velutina*, *Echinochloa pyramidalis*, *Panicum miliacum*, *P. maximum*, *Ischaemum pilosum*, *Dinebra retroflexa*, *Digiteria sanguinalis*, *Panicum trypheron*, *P. antidotale*, *Bothriochloa ischaemum*, *Elettaria cardamomum* (Rao *et al.*, 1965).

The virus disease is very common in India.

Ragi Mosaic (Leafhopper transmitted)

Govindu, *et al.* (1966) were the first to report a disease of complex nature of Ragi in Karnataka State (Mysore), India which involves both a fungus and a virus; the latter caused mosaic symptoms on the leaves (Govindu and Shivanandappa 1967). Yaraguntaiah and Keshavamurthy (1969), Keshavamurthy and Yaraguntaiah (1969) found that the disease was neither sap transmissible nor was it transmissible by any of the aphid species tested. The disease was, transmitted however, by the Delphacid *Sogatella* sp.

Therefore, the virus differs in mode of transmission from the

viruses reported by Joshi *et al.* (1966) and Rao *et al.* (1965). The virus reported by Joshi *et al.* (1966) was transmissible by sap and that of Rao *et al.*, by both sap and aphids. Yaranguntaiah and Govindu (1968) conducted varietal screening for Ragi mosaic virus-Delhi Isolate.

The disease is widespread in India.

BAJRA (*Pennisetum typhoides*)

Mosaic

Symptoms: The affected plants are smaller in size, pale in appearance and their leaves exhibit severe mosaic mottling.

Transmission: The aphid *Rhopalosiphum maidis* is the vector. The virus is readily transmissible when the juice is extracted in phosphate buffer at pH 7.0.

Properties: The virus has a thermal death point of 50 to 52°C, dilution end point between 1:500 to 1:1000, longevity *in vitro* at room temperature eight to sixteen hours at 7°C for a week.

Host range: It infects Bajra varieties H.B.1, H.B.4 and Hybrid 23A, K 559, rice varieties TN1 and TR-8, maize, jowar, ragi, *Setaria italica*, *Panicum miliare*, *P. miliaceum*, *Urochloa stolonifera*, *Chrysopogen montanus* and *Themeda quadrivalvis* producing mosaic symptoms. The virus does not infect sugarcane, Johnson grass, wheat and barley (Seth *et al.*, 1971).

The disease has been reported from India.

Streak

Symptoms: The diseased plants are chlorotic and stunted and leaves show long chlorotic stripes running along the whole length of the leaves. The affected plants produce poorly filled heads. The early infected plants are very much stunted and develop almost empty heads (Seth *et al.*, 1972).

Transmission: The disease is not transmitted through sap. The virus is transmitted to bajra by the commonly occurring leaf hopper, *Cicadulina mbila*.

Host range: It infects maize, sorghum, ragi, wheat, barley, oat, *Panicum miliaceum*, *Setaria italica* and *S. verticillaria* but not sugarcane (Seth *et al.*, 1972).

The virus has been designated as Pennisetum strain of maize streak virus and its disease is prevalent in India.

Chlorosis of Sorghum

Symptoms: The diseased plants are characterised by chlorotic leaves. Early symptoms of the disease are the broken longitudinal chlorotic bands in the laminae of young leaves running parallel to the midrib. Later the entire whorl of young leaves turn chlorotic. The leaves are much reduced in size and bunched together at the apex on account of retarded growth of internodes. Also the leaves of diseased plants are smooth and malleable unlike those of healthy plants, which are rough and brittle in texture. The virus induces early flowering of infected plants and stimulates prolific growth of nodal buds and tillers from near the ground level which remain dwarf, chlorotic and sterile.

The diseased plant usually bears malformed, twisted and poorly developed earheads.

Transmission: The virus is transmitted by *Perigrinus maidis* and incidence varies from 6 to 7 per cent in the rainy season and fifteen to ninety per cent in winter (Capoor *et al.*, 1968).

Host range: Three hundred ninety-two varieties of sorghum were found infected in field and the virus has been transmitted experimentally to *Canna orientalis*, *Cynodon dactylon*, *Euchlaena annulatum*, *Denbra retroflexa*, *Euchlaena mexicana*, *Pennisetum typhoides*, *Saccharum officinarum* and *Zea mays*.

The disease in sorghum is considered to be caused by a distinct virus designated 'chlorosis of sorghum' assigned binomial *Fractiliena sorghii* sp. and placed in the genus *Fractilina* of McKinney (1944).

The virus disease is found in India and USA.

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Oil Seeds

GROUNDNUT (*Arachis hypogaea*)

Mosaic

Symptoms: The symptoms are first visible on the young unfolding leaves which show a yellow discolouration of the veins. Later, the diseased leaves show yellow streaks along the lateral veins followed by development of chlorosis at the leaf tips. In case of early infection the plant develops a bushy appearance.

The disease has been recorded from Java.

Transmission: The virus is not sap transmissible and the vector is the leaf hopper *Orosius argentatus* (Smith, 1957).[~]

A mosaic disease of groundnut has also been described from India (Nariani and Dhingra, 1963).

Symptoms: The leaves of the diseased plants show circular chlorotic rings in early stages of infection followed by the appearance of dark green blister-like chlorotic patches. More often the leaves are malformed, puckered with narrow and pointed tips. Reduction in leaf lamina to form filiform leaves is a common feature. In later stages, the cluster of such malformed leaves give the plant a bunchy top appearance.

Transmission: The disease is graft transmissible and could not be transmitted by sap inoculation, through seed or soil or by *Aphis craccivora*, *A. gossypii*, *Bemisia tabaci*, *Empoasca devastans*, *Orosius* sp. and a number of unidentified leaf hoppers and beetles. Chenulu *et al.* (1966) reported yield losses of groundnut mosaic ranging from 29–100 per cent in kernel weight and 22–97 per cent in pod weight depending on disease intensity.

Mottle

Symptoms: The leaves of affected plants show leaf mottling, upward curling of the leaflets and depression of interveinal tissue. This disease is reported from Bulgaria, Venezuela and Ghana.

Transmission: The virus is transmitted mechanically and by the aphids, *Myzus persicae* and *Aphis craccivora* (Kuhn, 1964, Harold and Munz, 1969).

Properties: The thermal inactivation of the virus is found to be 60°C. The dilution end point is 10^{-8} and longevity *in vitro* is twenty-four hours at 25°C (Kuhn, 1965).

The virus has particle size of 812×15 nm (Harold and Munz, 1969) and 763×15 nm (Schmidt and Schmelzer, 1966).

Host range: Local lesions are induced on the primary leaves of some bean (*Phaseolus vulgaris*) varieties and mosaic on Cassia.

Ring Spot

Symptoms: The disease, prevalent in S. Africa, is characterised by well defined concentric rings on leaves. One or two reddish brown rings develop around a reddish brown central spot mostly non-marginal. The ring spot size varies from 3-5 mm in diameter (Kuhn *et al.*, 1964).

Transmission: The virus is sap transmissible (Klessner, 1966).

Host range: It includes many varieties of groundnuts and other species of legumes and also petunia and three species of *Nicotiana* (Klessner, 1966).

Rosette

Symptoms: The first sign of infection consists of a faint mottling of the youngest leaflets, together with slight vein necrosis. The next leaf is predominantly of a pale yellow colour upon which the veins form a green net work. The latter leaves are a little reduced in size and usually have distorted leaflets; the petiole and rachis are shortened. The whole plant may be a little more than a close tuft of small leaves forming a 'cushion' thus making the plant rosetted.

In some plants, however, the leaflets may exhibit a characteristic and pronounced mosaic pattern. The rosetted plant may flower, but few of the pegs make growth and none bear seed (Storey and Bottomley, 1928). Hull and Adams (1968) differentiated two main types, chlorotic rosette and green rosette and showed that rosette

is a complex of two viruses one of which is not aphid transmissible without the presence of a second virus called 'helper' virus.

Virus has a wide distribution over tropical and sub-tropical regions of Africa. It has been recorded from Gambia (Hayes, 1932), Uganda (Hansford and Hayes, 1940) and also from Senegal, Madagascar, Sierra Leone, Tanganyika, Java and India.

Transmission: Both the viruses of the rosette complex appear to be mechanically transmissible but the transmission is aided by use of buffer consisting of magnesium or sodium bentonite, one per cent potassium hydrogen phosphate and 0.01 M DIECA, pH 7.3 (Hull and Adams, 1968). The vectors are *Aphis craccivora* and *A. gossypii* (Watson and Okuyama, 1967).

Properties: The thermal inactivation point is 50°C for ten minutes exposure, the dilution end-point is 1:10 to 1:100 and the longevity *in vitro* is one week at 18°C and four-weeks at -20°C. The virus particles are isometric and measure 25 to 28 nm in diameter (Okuyama and Watson, 1966).

Host range: The virus induces local lesions on *Chenopodium amaranticolor*, *C. hybridum* and *C. quinoa* and infects *Trifolium incarnatum*, *T. repens*, *Nicotiana clevelandii* and *N. rustica* (Okuyama and Watson, 1966).

The groundnut rosette was first reported in India from Tamilnadu as a 'clump' disease (Sundararaman, 1928). Singh and Gupta (1966, 1968) reported three main types of symptoms, namely, normal rosette, mosaic and mottling from Rajasthan and have reported transmission to several other hosts. Kousalaya *et al.* (1971) reported that the groundnut rosette virus was spread by *A. craccivora* in Tamil Nadu in groups around the infected plants and there was a positive correlation between the aphid infestation and the incidence of the disease.

Control: The best means of controlling the rosette disease would be the introduction of resistant or tolerant varieties, proper spacing in the field and use of systemic insecticides. Close spacing has been found to reduce disease incidence. Spray trials with 0.5 per cent Schradan gave promising results in controlling the aphid vector and consequent check of the disease spread. Variety Mwitunde has shown lowest incidence of the disease and gives higher yields.

‘BUNCHY TOP’, ‘CHLOROSIS’ AND ‘RING MOTTLE’

Sharma (1966) identified three diseases of groundnut occurring in India.

Bunchy Top

Symptoms: Initial symptoms consisted of chlorotic mottle. The disease progressed rapidly, newly formed leaves were reduced in size and malformed; internodes were shortened and showed a range of faint mottle and chlorotic spotting. The terminal symptoms appeared in the form of complete suppression of internodes, partial sterility, mottled and cupped leaves. The infected plants were stunted and erect.

Transmission: Virus was graft transmissible but was not transmitted through sap or the insects tried. The virus proved to be seed borne.

Through wedge grafts *Desmodium diffusum* and *Alysicarpus longifolius* were successfully infected.

Chlorosis

Symptoms: Characteristic symptoms of the disease appeared in the form of marked chlorosis and severe stunting. The plants developed dwarf axillary shoots with stunted yellow leaves, often with red and discoloured margins. Older leaves were shed away. Young plants artificially infected with the virus did not develop flowers.

Transmission: Virus was readily transmitted through wedge grafts but was sparingly transmitted through sap inoculations (8 per cent transmission). Black bean aphid *Aphis craccivora* transmitted the virus in a persistent manner and retained the virus upto fifteen days. The virus was carried in seed collected from artificially infected plants. *Desmodium diffusum* and *Alysicarpus longifolius* were infected by grafting.

Ring Mottle

Symptoms: On naturally infected plants the only symptoms were numerous concentric chlorotic rings dispersed in faint mottle on the leaves. The rings later turned necrotic dark brown.

Transmission: Virus was readily graft transmissible. All attempts at sap transmission were negative. *Aphis craccivora* and *Orosius* sp. failed to transmit the virus from groundnut to groundnut. Virus was found to be seed borne upto three per cent in seeds collected from naturally infected plants.

Desmodium diffusum and *Alysicarpus longifolius* contracted infection through grafting.

The three viruses bunchy top, chlorosis and ring mottle did not offer cross protection to each other. Hence they do not seem to be related to each other.

Spotted Wilt or Chlorosis

Holmes *et al.* (1961) identified 'chlorosis' as being caused by tomato spotted with virus.

Symptoms: Symptoms consist of distinct ring spot and line pattern on inoculated leaves. Newly emerged leaves are small and round or pinched inwards and rugose. They show various patterns of mottling and small ring spots. Necrotic spots and irregularly shaped lesions develop on the leaves and along the petioles. Severely diseased leaves abscise. Later necrotic streaks develop on the stems. Internodes are reduced in size and short axillary shoots develop distorted leaves. This results in a bunched appearance. Flowers tend to be reduced in number. The disease has been reported from Brazil, South Africa and Queensland.

Transmission: The disease is not seed transmitted although seeds develop necrotic lesions and are malformed. Thrips are recognised to be the vectors of the virus, the efficient one being *Thrips tabaci*.

Properties: The longevity *in vitro* of the virus is less than five hours in sap extracted from *Nicotiana glutinosa*.

Host range: There are a large number of alternate hosts of the virus including several typical weeds, e.g., *Bidens pilosa*, *Physalis minima*, *Tagetes minuta*, *Erigeron bonariensis* and *Trifolium subterraneum*.

Bud Blight

Symptoms: Symptoms included ring spot pattern on the topmost expanding leaves of one or other shoot of the affected plants. The ring spots had green islands surrounded by a ring of yellow tissue. Later, the shoot just near the growing tip became necrotic and dried. The necrosis, however, might kill the whole branch, but some branches of the affected plant remained alive with all shoot buds blighted. Axillary buds sprouted from the living branches of the plant but the leaves were very much reduced in size, showed mosaic pattern, curling, cupping, puckering and continue to survive till late in the season even after the healthy plants matured and harvested. The pods produced by the affected plants were

distorted, small in size, shining with a deep constriction in the middle. Further flowering and pod formation by the affected plants was stopped.

- Chohan (1972) reported the bud blight disease of groundnut from Punjab which manifested itself after about a month after sowing.

Transmission: Bud blight is not transmitted by seed or aphids. It is graft transmitted and with sap extracted in buffer and 0.5 per cent sodium sulphite (Chohan, 1972).

Properties: It is inactivated at 40°C for ten minutes.

Host range: It causes local lesions in groundnut, guar, broadbean, mash, cowpea, bitter gourd, muskmelon and longme on.

On the basis of the results reported it has been suggested that the bud blight of groundnut in Punjab is caused by the tomato spotted wilt virus.

Capoor (1964) reported a disease from Poona showing similar symptoms called 'shoot necrosis' which was attributed to a fungus *Fusarium solani* with the rosette virus.

Reddy *et al.* (1968) reported a bud necrosis from Andhra Pradesh which was graft transmissible. The groundnut varieties 'Spanish Improved' and 'Asiriya Mwitunde' were highly susceptible.

Symptoms: Necrosis of terminal buds of plants is followed by saprophytic fungal invasion and proliferation of decaying buds, resulting in shoots with small, mottled leaves. In advanced cases the whole plant remained bushy, stunted and occasionally died.

Transmission: The disease is graft transmissible.

According to the authors, *Fusarium* and *Cephalosporium* sp. were isolated from necrotic buds but these were mostly saprophytic. Recently *Phoma glomerata* has also been proved to be associated with the bud blight virus disease (Dhanju and Chohan, 1974).

Chlorotic Spot

Symptoms: Disease is characterised by development of chlorotic leaf spots in terminal leaves of axillary shoots followed by appearance of mottle.

The disease was reported by Hargopal and Nayudu (1971) from Tirupati, India.

Transmission: The virus is sap transmissible. Transmission can be improved by addition of buffers to the inoculum.

Properties: The virus has thermal inactivation point of 36–40°C. The dilution end-point ranges from 1/100 to 1/1000 and longevity at 33°C, for two hours.

Host range: Following plants were systemically infected besides groundnut under experimental conditions: *Clitoria ternata*, *Crotalaria serices*, *C. juncea*, *Phaseolus vulgaris*, *Cassia occidentalis*, *Canavalia ensiformis*, soybean, pea and *Sesbania* sp.

MUSTARD (*Brassica* sp.)

CHINESE SARSON (*Brassica juncea*) var. *Rugosa*

Mosaic

Symptoms: The diseased plants show vein clearing, vein banding, mottling and severe puckering of the leaves. The affected plants are stunted and flowering is either absent or scanty, producing only a few poorly filled and shrivelled fruits. In advance stages of infection the stem and fruits also show distinct mottling. The disease incidence is fairly high resulting in considerable loss in yield.

Transmission: The virus is found to be readily transmissible by sap inoculation and also by *Aphis gossypii*. *Brevicoryne brassicae* and *Myzus persicae* (Azad and Sehgal, 1959). *A. rumicis* also transmits *B. juncea* mosaic virus in a non-persistent manner. (Azad *et al.*, 1963).

Properties: The virus is inactivated between 52–55°C by ten minutes exposure and has a dilution end-point of 1:1000–1:3,000 and infectivity *in vitro* for about two days.

Host range: The virus can be transmitted to a variety of plants in cruciferae through the aphid vector *B. brassicae*. It is transmitted by sap inoculation, causing systemic infection on *Z. elegans* and local lesions on *N. tabacum*.

The virus belongs to turnip virus I group.

RAI (*Brassica juncea*)

Mosaic

Symptoms: The affected leaves show characteristic mosaic symptoms and deformation of leaf lamina. The diseased plants become stunted.

Transmission: The causal virus is sap transmissible. It is not seed borne. *Myzus persicae* and *Lipaphis erysimi* transmitted the virus.

Properties: The virus is inactivated by heating at 68° C for ten minutes. Dilution end point ranges between 1/5000–1/7000 and longevity *in vitro* for twelve days at 22° C.

Host range: *Brassica juncea*, *B. rugosa* and *Chenopodium quinoa* were found to be susceptible (Sharma, 1973).

SAFFLOWER (*Carthamus tinctorius*)

Mosaic

Symptoms: Symptoms of the disease consist of a light and dark green mosaic on the leaves and involucral bracts. In a few plants, the primary leaves are produced forming a rosette of leaves exhibiting mosaic mottling and from the centre of which the axis bearing the secondary leaves is produced. The affected plants though developed to maturity, seeds produced by these plants are poor in quality.

Transmission: The virus is transmitted by sap and by *Myzus persicae*, *Aphis gossypii* and *A. craccivora*.

Properties: The virus can withstand heating for ten minutes at 69° C but not at 70° C, retains infectivity upto a dilution of 1:1,000 and storage for twenty-five hours at room temperature (26–30° C).

Host range: The host range of the virus is very wide. The virus is found to infect members of the families; Compositae, Solanaceae, Amaranthaceae, Balsaminaceae, Cucurbitaceae, Apocynaceae, Umbelliferae, Leguminosae, Chenopodiaceae, Commelinaceae and Graminae.

Based on the above studies, the virus is identified to be a strain of Cucumber mosaic virus (CMV) (Thangamani *et al.*, 1970).

More or less similar symptoms have been reported to be observed by Klisiewicz (1962) on safflower in California and he identified the causal virus to be cucumber mosaic virus. Muller (1959) reported the occurrence of a safflower mosaic virus from California, Arizona and Utah. A mosaic occurring in leaves and bracts of safflower grown in Israel is found by Nitzany (1960) to be caused by CMV.

Mosaic disease caused by a different virus has been described from Delhi by Chenulu *et al.* (1971).

Symptoms: The affected plants develop prominent vein clearing after twenty days' inoculation. This is soon accompanied by pronounced mottling, alternate light and dark green patches with occasional blistering and distortion of the leaves.

Transmission: The virus is mechanically transmitted. The virus is also transmitted to the extent of three to four per cent through true seed. *Myzus persicae* and *Aphis gossypii* are the vectors reported.

Properties: The virus inactivates by exposure at 62-65°C for ten minutes. The dilution end-point of the virus ranges between 1:2000 to 1:5000. The virus in extracts from infected plants remained infective by storage at room temperature for five to six days. The virus particles are found to be 260 nm long and 18 nm wide in shadowed preparations.

Host range: The causal virus has a fairly wide host range infecting several plant species of Chenopodiaceae, Compositae, Cucurbitaceae, Leguminosae, Solanaceae and Amaranthaceae.

The virus belongs to tobacco mosaic virus group. It has been concluded that the disease reported here is caused by a rod shaped virus which resembles in symptoms, transmission, host range and properties, a virus earlier described by Bayden and Nixon (1951).

SESAMUM (*Sesamum orientale*)

Leaf Curl

Symptoms: The virus causes severe downward curling of the leaves. Leaves become leathery and show thickening of veins on the underside of the leaves. Severely affected plants remain stunted and bear few fruits, if the infection occurs in the sixth week after sowing or at a period earlier to this, the loss in crop yield is heavier (Sahambi, 1958).

Transmission: The virus is transmitted through the agency of whitefly *Bemisia tabaci*.

Control: Early appearance of the disease results in considerable reduction in yield. Weekly spraying of the crop with solidol, E 605 and Ekatox 20 W.P. results in about two weeks delay in the appearance of the disease and increases the yield considerably (Sahambi, 1958).

Mosaic

Symptoms: The virus causes conspicuous chlorotic areas of irregular shapes on the leaf lamina. Interveneal areas are mostly yellow. The top portions and margins are yellowish to complete yellow the basal portions remaining green. The young developed leaves are completely yellow in colour. With the advance of the disease the top leaves become gradually smaller in size and plants cease to grow (Gangopadhyay, 1967).

Transmission: The virus is graft and sap transmissible. Further information is lacking.

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Ornamentals

BOUGAINVILLEA (*Bougainvillea spectabilis*)

Mosaic

Symptoms: The disease is characterised by the development of distinct interveinal chlorosis followed by general chlorosis and reduction in leaf size. It occurs in India (Garga, 1966).

Transmission: The disease is neither transmitted by sap inoculation nor by dodder (*Cuscuta reflexa*). The virus is transmitted by grafting as well as by two species of aphids, namely, *Myzus persicae* and *Lipaphis erysimi* Kalt.

Host range: The host range of the virus is restricted to *Bougainvillea* only (Garga, 1966).

BUTTER CUP (*Ranunculus asiaticus*)

Mosaic

Symptoms: In the early stages, the leaves of the affected plants show mosaic mottling comprising of light and dark green patches. The severely infected plants are stunted with colour breaking in flowers. The plants mature faster than the healthy plants. It has been reported from India.

Transmission: The disease is readily sap transmissible. It is also transmitted by aphid vectors, namely, *Aphis fabae*, *A. gossypii*, *A. nerii*, *Lipaphis erysimi*, *Macrosiphonella sonbornii*, *Myzus persicae* and *Rhopalosiphum maidis*. *M. persicae* transmitted the virus in a non-persistent manner from *Ranunculus* to *N. glutinosa*.

Properties: The virus is inactivated by ten minute exposure of the infectious extract at 75°C but not at 70°C. It is inactivated when

the infective extract is diluted more than 1:1000. It has long flexuous particles of 800 nm size.

Host range: The host range is restricted to delphinium, petunia, tobacco and chenopodium (Padma *et al.*, 1972a).

CANNA INDICA

Mosaic

Symptoms: The affected leaves exhibit irregular, pale yellow stripes running parallel with the veins and extending from the midrib to the margin. The affected foliage is more or less wrinkled and curled. The chlorotic areas on the leaves often turn rusty brown. The stem, sepals, and petals bear yellowish bands while the fruit displays indistinct mottling (Ocfemia *et al.*, 1942). The disease is reported from Philippines, Japan, USA and India.

Transmission: The virus is transmitted by sap with difficulty. But it is transmitted by several aphids, namely, *Myzus persicae*. *Aphis gossypii*, *A. maidis*, *Macrosiphum solanifolii* and *Myzus circumflexus* and is of non-persistent type. There is no evidence of seed transmission.

Host range: Species of *Canna* susceptible to the virus are *Canna dauca* and *C. generalis*. Ocfemic *et al.* (1942) found that *Musa textilis* is also susceptible to the virus.

Mottle

The virus is studied and described by Dutta Gupta (1974).

Symptoms: First symptoms appear on young leaves in the form of vein clearing which are followed by characteristic mosaic mottling and blistering. Mature leaves show dark green and light chlorotic stripes running parallel to the veins. The green stripes become faint gradually but the yellow chlorotic stripes become more prominent and dark yellow to brown necrosis is observed on them. Elongated spindle shaped and plate like inclusion bodies are observed in the epidermal peelings.

Transmission: The virus is transmissible by sap inoculation as also by injecting rhizomes with infective sap, the latter method giving 100 per cent infection. Four species of aphids, namely, *Aphis gossypii*, *A. rumicis*, *Myzus persicae* and *Rhopalosiphum maidis* transmitted the virus.

The virus vector relationship is of the non-persistent type.

Host range: The virus is transmissible to *Zea mays*, *Hordeum vulgare*, *Pennisetum typhoides*, *Gomphrena globosa*, *Petunia hybrids*, *Vinca rosea*, *Colocasia antiquorum*, *Zinnia elegans*, *Phaseolus lunatus*, *Gisna sincusis*, *Nicotiana tabacum* and *N. glutinosa*.

Physical properties: The virus has a thermal inactivation point between 65–70°C, a dilution end point of 1 : 7500 to 1 : 8000 and lingering *in vitro* of 74 days at room temperature (28–32°C). The virus particles are spherical with a mean diameter of 27nm.

The virus is serologically related to cowpea mosaic virus of Chenulu *et al.* (1968).

CHRYSANTHEMUM (*Chrysanthemum indicum*)

Aspermy

Symptoms: Mild mosaic mottling with marked distortion of the flowers. The flowers borne on the diseased plants are small in size and are distorted with irregular curlings and waviness of the ray florets. The central disc florets remain green and stunted. Line pattern with diffuse chlorotic mottling appear on the new leaves.

Transmission: The disease is sap transmissible. *M. persicae* acts as the vector of the virus in India (Sastry, 1964).

Properties: The virus has thermal inactivation point of 60°C and dilution end point of 1:100; and the longevity *in vitro* is less than six hours.

DAHLIA

Mosaic

Symptoms: The disease is characterised by the development of 'vein banding'. The normal green colour develops irregularly in the mosaic leaf bands adjacent to the midrib, branch veins remaining yellowish green or pale green when the remainder of the leaf reached normal colour. As the affected leaves become older the discoloured areas tend to approach the normal green in the remainder of the leaf and the colour pattern may become masked. Vein banding is the most reliable diagnostic symptom of Dahlia mosaic.

Transmission: The disease is sap transmissible (Lepine *et al.*, 1951). The insect vectors are aphids, the most important one being *Myzus persicae*. Other species are *Aphis fabae*,

A. gossypii, *M. convolvuli* and *M. acrosiphugei*.

Properties: The thermal inactivation point is between 85°C and 90°C, the dilution end point is around 1:3000 and the longevity *in vitro* is between twenty-eight and thirty-five days. The virus retains infectivity in drying leaves for ten days but not for fourteen days.

The virus is serologically related to cauliflower mosaic virus, but not to viruses of arabis mosaic, tomato black ring and broad bean mottle (Brunt, 1966). The virus has isometric particles measuring about 50 nm in diameter. The particles are very similar in morphology, size and density to those of cauliflower mosaic virus (Brunt, 1971).

Host range: It is transmitted to *Zinnia elegans* and *Verbezcina encelioides* through sap (Brierley and Smith, 1950).

HELPTERUM (*Acroclinium roseum*)

Mosaic

Symptoms: In India, the diseased plants exhibit stunted growth harrowing of the leaves and presence of dark green patches on the leaf lamina (Vashisth, 1963).

Transmission: The disease is readily transmitted mechanically and is also transmitted by three aphid species, namely, *Aphis gossypii*, *Brevicoryne brassicae* and *Myzus persicae* (Vashisth, 1963).

Host range: The disease is readily transmitted to Chinese sarson, *Brassica niara*, *Brassica juncea* var. T 144m, *B. chinensis*, but is not transmitted to the non-cruciferous hosts such as beet, bhindi, cowpea, cucumber, spinach and turkish tobacco variety symarna (Vashisth, 1963).

HIBISCUS MANIHOT

Mosaic

Symptoms: The disease is characterised by development of mild mosaic and vein clearing on young leaves. The mosaic symptoms become marked as the leaves mature (Van Valsen, 1967). The disease is reported from Papua and New Guinea.

Transmission: The virus is transmitted mechanically but not by seed, sap or insects (Van Valsen, 1967).

Host range: The host range of the virus is restricted to *Hibiscus* spp. only (Van Valsen, 1967).

It does not appear to be related to any virus previously described on *Hibiscus*.

HIPPEASTRUM sp.

Mosaic

In India, a mosaic disease on *Hippeastrum* plants has been observed since last five to six years.

Symptoms: The affected plants show elongated dark and green stripes on leaves and flower stalks. The leaves or flowers are not deformed. In severe cases of infection the plants remain stunted and do not bear quality blooms.

Transmission: The disease is sap transmissible and it is spreading in nature through infected bulbs. Further studies on host-range, insect transmission, histopathology, particle morphology and control of the disease by heat and chemotherapy are in progress at the IARI (Padma and Raychaudhuri, 1976).

JASMINE (*Jasminum sambac*)

Chlorotic Ring Spot

Symptoms: The disease is characterised by development of typical mosaic symptoms consisting of yellow chlorotic spots or areas of varying size and shape. The spots are intermingled with the normal green colour of the leaves. Frequently yellow coloured rings having green centre appear on some of the leaves along with mosaic pattern. The rings are generally single and measure 2.5 mm to 6.00 mm in diameter and are not delimited by the veins (Wilson, 1972). The disease is reported from India.

Transmission: The virus is not sap transmissible. However, it could be easily transmitted by inarch grafting.

The virus is also transmitted by the whitefly (*Bemisia tabaci*) (Wilson, 1972).

JATROPHA GOSSYPIIFOLIA

Mosaic

Symptoms: The disease is characterised by the development of yellow blotches delimited by veins on the affected leaves (Bird, 1957).

Transmission: The virus is transmissible neither by sap nor through seeds but is easily transmitted through grafting and by *Bemisia tabaci*.

The virus appears to be a strain of tobacco leaf curl virus.

JATROPHA CURCAS

Leaf Distortion

Symptoms: The disease is characterised by marked reduction of leaf size, upward rolling of the margins, prominent puckering of the leaf margin and the main veins; and occurs in India.

Transmission: The disease is transmitted by grafting (Garga, 1961).

MARIGOLD (*Tagetes erecta*)

Mosaic

Symptoms: The leaves of the affected plants develop mosaic mottling at early stages of infection and later leaf lamina is considerably reduced and distorted. The diseased plants remain stunted and produce poorly developed flowers. (Sang and Varma 1974). The virus is common in Northern India.

Transmission: The virus is transmitted by sap and by aphids *Aphis craccivora*, *A. gossypii*, *A. pseudobrassicae* and *Myzus persicae* in a stylet borne manner. *A. gossypii* could acquire the virus through stretched Parafilm.

Properties: The thermal inactivation point of the virus is 55°C, dilution end point 1:6000 and ageing *in vitro* at 30+2°C for more than four days and at 7°C for more than seven days. The concentrated preparations obtained by differential centrifugation contained particles of 28nm diameter.

Host range: The virus infected twenty-three plant species belonging to families Leguminosae, Cucurbitaceae, Chenopodiaceae, Compositae, Amaranthaceae, Solanaceae, Scrophulariaceae and Polemoniaceae. It produces bright mosaic in systemically infected leaves of *Nicotiana tabacum* and distinct chlorotic lesions turning necrotic on inoculated leaves of *Chenopodium amaranticolor* within a week after inoculation.

The virus is a member of cucumis virus group.

MALAVAVISCUS (*Malvaviscus arboreus*)**Leaf Curl**

Symptoms: Affected leaves show faint leaf mottling and chlorosis in early stages of disease development, later vein thickening becomes pronounced. Leaf size is occasionally much reduced with enations on under surface. Puckering of leaf is a common feature and the margins of severely affected leaves frequently curled upwards. The disease is reported from India.

Transmission: The virus is not sap transmissible but could be transmitted by wedge-grafting and by the vector *Bemisia tabaci*.

Host range: The virus infects *Hibiscus rosa-sinensis*, *Abelmoschus esculentus*, *A. tuberculatus* and *A. manihot* (Mukherjee and Raychaudhuri, 1964) and is believed to be caused by leaf curl virus of *H. rosa-sinensis*.

NASTURIUM (*Tropaeolum majus*)**Mosaic**

Symptoms: The virus causes mottling, vein banding and crinkling of the leaves. In severe cases of infection generalised stunting is seen (Silverschmidt, 1953). The disease is reported from USA and Brazil.

Transmission: The virus is readily transmitted by mechanical means and has several species of aphid vectors *M. persicae*, *M. circumflexa*, *Aphis rumicis* L., *A. gerruginea striata* and *Rhopalosiphum prunifoliae* (Jensen, 1950).

Properties: The thermal inactivation point of the Brazilian nasturtium virus is found to be 58°C, dilution end point 1:100, and longevity *in vitro* twenty-four hours at room temperature.

Ring Spot

Symptoms: The disease manifests itself as bright ring spots scattered on the leaf surface. Severely affected plants show mosaic mottling, the leaves become crinkled and distorted. The whole plant is stunted (Smith, 1950).

Transmission: The virus is transmitted by *M. persicae* and *Brevicoryne brassicae* in non-persistent manner but is not transmitted by *Macrosiphum pisi*.

Host range: The virus is transmitted to eight species of Solanaceae

and Leguminosae (Bhargava and Joshi, 1959).

A sap inoculable ring spot virus was isolated from double *Tropaeolum* plants collected from Simla Hills by Mishra (personal communication). It resembles the tobacco ring spot virus in host-range, physical properties, serological relationship and structure and composition of Capside though transmitted by aphids.

ORCHID (*Cattleya* sp.)

Mosaic

Symptoms: The symptoms of the disease are manifested as colour 'break' in the flower, whereby the petals show mottling together with some distortion. The leaves show mosaic mottling and malformation.

Transmission: The virus is transmitted through sap and also by *Myzus persicae* (Jenson, 1949).

Properties: The virus has rod-shaped particles measuring about 400 nm \times 18 nm (Gold and Jensen, 1952).

PANAX (*Nothopanax quilfolei*)

Ring Spot

Symptoms: The disease initiates development of concentric rings on the leaves, premature leaf shedding and stunting of the plants. The ring spots start as tiny chlorotic areas and as the disease advances, large number of concentric rings are formed. With time the outer edges of the rings become purplish brown. Shedding of the leaflets is not observed although occasionally nearly complete defoliation has been observed in individual plants (Aragaki *et al.*, 1953).

Transmission: The virus is transmitted by grafting and inarching but not sap transmissible.

The virus has two aphid vectors namely *Myzus persicae* and *Aphis gossypii*. It has been reported from Hawaii.

PERIWINKLE (*Vinca rosea*)

Mosaic

Symptoms: The disease manifests itself as severe mosaic mottling associated with malformation of the leaf lamina.

The virus is reported from India.

Transmission: The virus is sap transmissible.

Properties: The thermal death-point is 50–55°C, the dilution-end point is between 1:800 and 1:900. The longevity *in vitro* at room temperature (27–40°C) and 7–10°C is found to be twenty-four to forty-eight hours, and five to six days respectively (Joshi and Raychaudhuri, 1964).

Host range: The virus is transmitted to *V. rosea*, *N. tabacum*, *N. tabacum* cv. White Burley, *N. glutinosa*, *N. rustica*, *Petunia hybrida*, *Cucumis sativus* and *C. melo* cv. *Utilissimus*. *Chrysanthemum morifolium* is symptomless carrier of the virus.

Leaf Curl

Symptoms: The disease is characterised by development of crumpling, curling and mosaic symptoms on the leaves, especially those at the apex of a branch. The flower size is reduced considerably. The discolouration of the leaves and fading of the flowers also occur in later stages of infection.

Transmission: The virus is graft transmissible but is not transmitted by dodder, whiteflies or sap inoculation (Bisht and Singh, 1964).

PETUNIA (*Petunia hybrida*)

Mosaic

Petunia mosaic is caused by several viruses, namely, tobacco mosaic virus, alfalfa mosaic virus, datura mosaic virus, tomato bushy stunt virus, potato virus X and cucumber mosaic virus either under natural conditions or by artificial inoculations.

Symptoms: The diseased plants show development of stunted growth and reduction in leaf size. The infected leaves exhibit a clear mosaic mottling comprising of irregular light green and dark green patches. The lower leaves exhibit the symptoms of vein banding. In some cases the leaves are malformed with blisters and raised areas.

Transmission: The virus seems to be a strain of either chilli mosaic virus reported by Jha and Raychaudhuri (1956) or Petunia yellow mottle reported by Rubio and Russell (1959). The virus is transmitted mechanically by sap inoculation. It is also transmitted by aphid vector, *Aphis gossypii* L. (Mishra and Chenulu, 1966).

A naturally occurring virus disease of *Petunia* showing dark green mosaic mottling has been reported by Rani *et al.* (1969) from UP State, India.

The virus is inactivated between 60–70°C, dilution beyond 1:10,000 and by storage for more than seventy-two hours at room temperature. The virus was found serologically alike to tobacco ring spot which is transmitted by nematodes.

A strain of cucumber mosaic virus causing a mosaic of *Petunia* is readily transmitted by *Myzus persicae*.

Properties: The thermal inactivation point of the virus is 60°C–70°C, dilution end-point is 1:200 and the virus remains infective in the expressed sap for ninety-six hours at room temperature 25–30°C.

Host range: It can be easily transmitted to *Nicotiana tabacum* cv. White Burley and Harrison's Special, *N. glutinosa*, *N. rustica*, *Capsicum annuum* and *Petunia hybrida*.

PHLOX (*Phlox drummondii*)

Mosaic

Symptoms: The disease shows up as slight vein clearing followed by light and dark green mosaic mottling of the leaves. There is no deformation or distortion of leaf lamina. The affected plants appear paler than normal. The symptoms are masked in summer (Khatri and Chenulu, 1966). The disease is reported from India.

Transmission: The virus is sap transmissible and also by *Aphis gossypii*. Single viruliferous aphid, *A. gossypii* is capable of transmitting the virus in a non-persistent manner (Rangaraju and Chenulu, 1968).

Properties: The virus is inactivated at 60°C (ten minutes exposure) at a dilution of 1:50 and after storage for sixteen hours at room temperature (25–33°C).

Host range: The virus is transmitted to phlox and several other plants, namely, *Nicotiana tabacum*, *N. glutinosa*, *Capsicum annuum*, *Solanum nigrum*, *Datura stramonium*, *Lycopersicon esculentum*, *Cucumis sativus*, *C. melo*, *Cucurbita pepo*, *Chenopodium amaranticolor*.

PRIMULA (*Primula* sp.)**Mosaic**

Symptoms: The diseased plants show mosaic pattern on the affected leaves (Nagaich and Giri, 1968).

Transmission: The virus is transmitted by sap inoculation and by *M. persicae* and *Aphis rumicis*.

Properties: The virus is inactivated by heating at 56–59°C at dilution of 1:600 and loses infectivity by storage for more than ninety-six hours.

It is found to be a strain of alfalfa mosaic virus.

Mottle

Symptoms: The affected plants are stunted and show colour break of flower and mottling of leaves.

Transmission: The virus is sap transmissible and is also transmitted by *M. persicae* and *A. gossypii*.

Properties: The virus is inactivated by heating at 60° C and loses infectivity by storage for two days at room temperature and at 4°C for five days. The dilution end point of the virus lies at 1:500. The virus particles measure 720 745 nm.

Host range: Of the thirty-eight plant species tested, only ten were found to be susceptible. The virus produced symptoms on *Chenopodium album*, *C. amaranticolor*, *Datura stramonium*, *Gomphrena globosa*, *Nicotiana glutinosa*, *N. rustica*, *N. tabacum*, and *Petunia alba* (Singh *et al.*, 1970).

The virus occurs naturally in *Primula malacoides* in India.

ROSE (*Rosa* sp.)**Yellow Mosaic**

Symptoms: The symptoms appear as yellow coloured bands adjacent to veins. Bands may be narrow or broad. In advanced stages these bands spread and cause clearing of the veins and veinlets. When the disease is fully developed the vein and veinlets become much prominent by developing more yellow colour and exhibit a sort of yellow net more on the green leaf surface. Yellowing of the veins and veinlets may sometimes be restricted to a portion of the leaflet and only to a few branches of the plants in certain cases.

Transmission: The virus is bud transmissible and is also transmitted by *B. tabaci*. The virus is reported from India.

Host range: The virus has been transmitted by grafting and budding to the following cultivated varieties of roses: Condesa de Sastago, Clovelly, Flaming Sunset G and more Jenny, Helen Traubet, Hilda, Lady Margaret Stewart, Mirandy, Miss George-Geary, Molly Skarman, Crawford, Opera, President Herbert Hoover, Radio and William Shean (Bhargava and Joshi, 1962).

ROSA BOURBIANA

Yellow Vein Mosaic

Symptoms: When the disease is fully developed the veins and veinlets become much more prominent by developing yellow colour. The leaves exhibit a sort of yellow net work on the green leaf surface. The plants flower normally, except for slight reduction in flower size followed by mild deformation.

Transmission: The virus is transmitted by grafting or budding. The disease is also transmitted by *Bemisia tabaci*.

The virus differs from rose mosaic streak virus and rose wilt virus (Sastry, 1966).

SHOEFLOWER (*Hibiscus rosa-sinensis*)

Line Pattern

Symptoms: The leaves of affected plants show yellow net vein symptoms. The virus is reported from South Africa.

Transmission: The virus is transmitted by grafting and is not readily sap transmissible. The evidence of aphid transmission is doubtful (Wolfswinkel, 1966).

Leaf Curl

Symptoms: The disease causes curling of the affected leaves to varying degree and development of enations on under surface takes place. The disease is widespread in India.

Transmission: The virus is transmissible through grafting as well as by *Bemisia tabaci*.

Host range: The virus is transmitted to *Althea rosea*, *Abelmoschus moschatus* by grafting (Vasudeva *et al.*, 1953) and to *Abelmoschus esculentus*, *A. tuberculatus* and *Hibiscus manihot* by whitefly.

SOAPWORT (*Saponaria* sp.)**Leaf Curl**

Symptoms: The affected plants are stunted in growth. The leaves are curled and small in size. Profuse irregular, granular outgrowths appear on the veins on the underside. The flowering is either scanty or absent (Azad, 1953).

Transmission: The disease is transmitted by grafting only.

SUNFLOWER (*Helianthus annuus*)**Mosaic**

Symptoms: Uppal (1933) reported the disease from Bombay. The virus causes a mosaic pattern, which is accompanied by ring spots or chlorotic spots which have a tendency to coalesce (Battu and Phatak, 1965). The disease is very much prevalent in India.

Transmission: The disease is transmitted by sap inoculation. Typical virus symptoms appeared in ten days at a temperature range of 29-41°C (Battu and Phatak, 1965).

Host range: The virus could not be transmitted to *Nicotiana tabacum*, *N. glutinosa* and *Capsicum annuum* (Battu and Phatak, 1965; Uppal, 1933) and is restricted to sunflower only.

THEVETIA NERRIFOLIA**Leaf Curl**

Symptoms: The diseased plants show curling of the leaf tips downwards and sideways, smalling of the leaves, asymmetry of lamina and waviness of the leaf margin followed by mild chlorosis near the midrib and veins.

Transmission: The virus is graft transmissible (Garga, 1953).

ZINNIA ELEGANS**Mosaic**

Symptoms: The disease is characterised by the appearance of light and dark green mottling of the foliage accompanied by vein clearing and green vein-banding. The affected plants are neither appreciably stunted nor the leaf size is reduced. The flowers appear normal but are fewer than those on the healthy

plants. The diseased plants are pale in appearance.

Transmission: The virus is sap transmissible. The aphids, *A. gossypii* and *Myzus persicae* also transmit the disease.

Properties: The thermal inactivation point is 50–53°C and the dilution end-point 1:100–1:200, longevity *in vitro* sixteen to eighteen hours at 10–18°C, five to six days, when stored at 7–9°C and pH stability between 4–10.

Host range: The virus produces systemic mosaic symptoms on *Nicotiana tabacum*, *N. glutinosa*, *Petunia hybrida*, *Cucumis sativus* and *Momordica charantia*.

The virus has been identified as a strain of *Cucumis virus 1* (Prasada and Raychaudhuri, 1961) occurring in India.

Leaf Curl

Symptoms: The disease is identified by the thickening of veins and veinlets on lower surface, curling of the leaves, dwarfing of the plants, distorted flowers with pale colour and partial sterility (Mathur, 1933).

Transmission: *Bemisia gossypiperda* is the vector of this disease. The disease is very widespread in India.

Mild Mottle Virus

Symptoms: The affected plants show vein clearing accompanied by mosaic mottling and a general paleness of the plants. Diseased plants bear fewer flowers than the healthy ones.

Transmission: The disease is easily transmissible by sap-inoculation.

Host range: The virus infects thirty-three plant species, belonging to eleven families, namely, *Amaranthaceae*, *Apocyanaceae*, *Cariaceae*, *Chenopodiaceae*, *Compositae*, *Cruciferae*, *Cucurbitaceae*, *Leguminosae*, *Malvaceae*, *Scrophulariaceae* and *Solanaceae*. It produces chlorotic and necrotic local lesions on *Chenopodium amaranticolor* and *C. album* respectively.

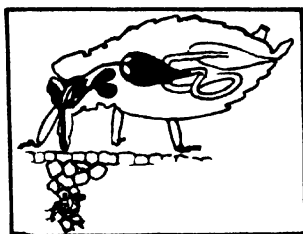
It causes systemic infection only on *Zinnia*. The virus has been designated as *Zinnia mild mottle virus* (Padma *et al.*, 1972b).

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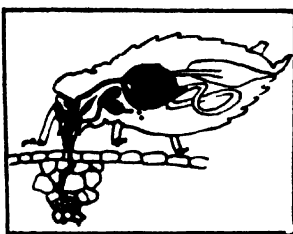
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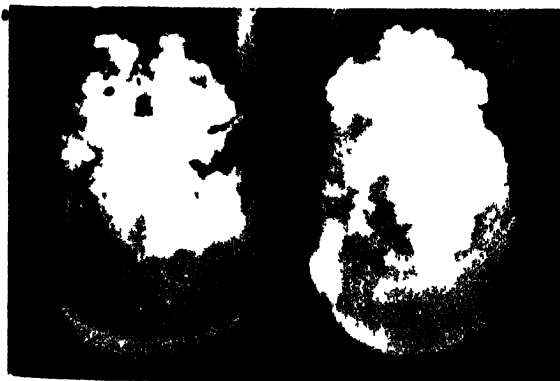
2 1 (a) Non Persistent Viruses



(b) Persistent Viruses



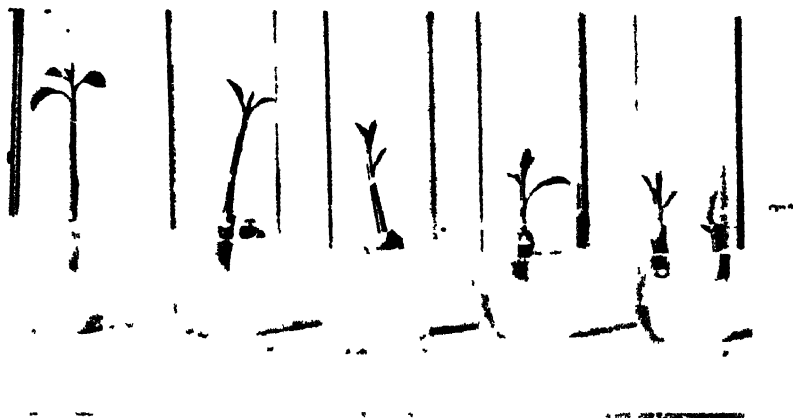
2 2 Leafhopper *Cicadulina mbila*
vector of bajra streak virus feeding
on bajra plant



7 1 Apple tumor virus in
tissue culture

Right Callus from tumor
tissue

Left Callus from healthy
tissue



7.2 Culture of Exocortis affected citrus bud wood on MS medium



7.3 Differentiation of shoots in stem callus tissue of *Citrus grandis*



7 4 Healthy sugarcane plant raised by meristem culture



10 2 Vein enation of maize



10 3 Vein enation of maize on wheat



10 4 Vein enation of maize on rice



10 5 Vein enation of maize on ragi



11 1 Yellow mosaic of jute



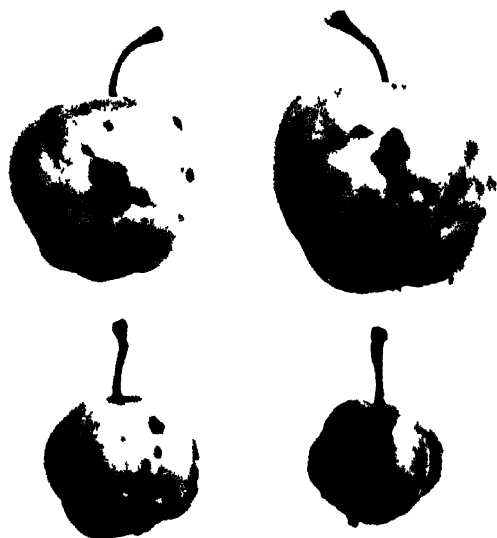
11 2 Sunnhemp mosaic



11.3 Electron micrograph of
Southern Sunn hemp mosaic virus



14.1 Apple mosaic



14 2 Apple star crack



14 3 Apricot mosaic



14 4 Banana mosaic



14.5 Banana bunchy top



14.6 Cape gooseberry
mosaic due to CMV



14 7 Cape gooseberry mosaic due to TMV



14 8 Cherry mosaic

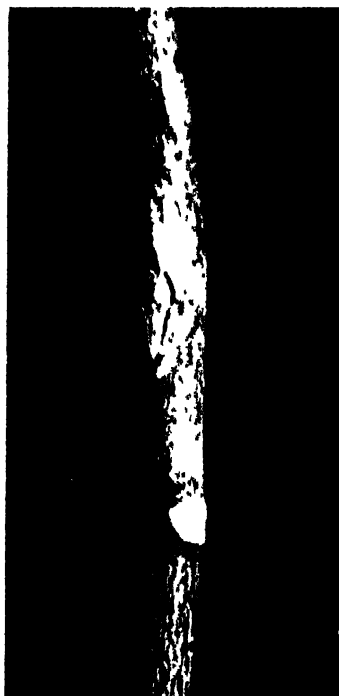
14 9 Tatter leaf of cherry



14 10 Leaves of lisbon lemon seedling showing spots when inoculated with lemon crinkly leaf virus



14.11 Leaves of eureka lemon affected with lemon crinkly leaf virus



14.12 Cracking of bark and elongated lesions by exocortis on Rangpur lime



14.13 Cracking of bark by exocortis on sweet lime



14.14 Exocortis affected grape fruit tree



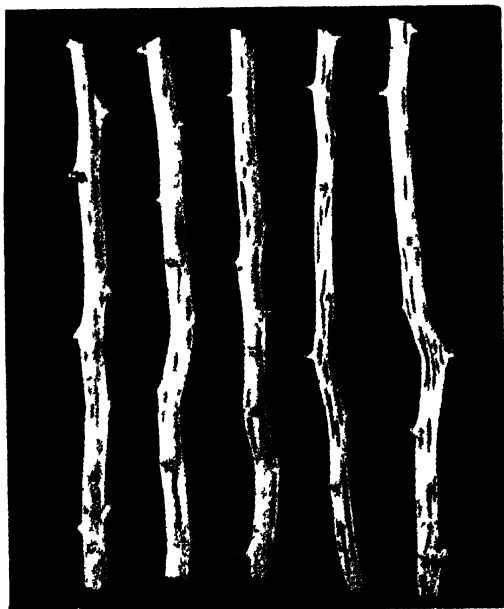
14 15 Citrus tristeza (affected sweet orange tree on sour orange rootstock on right)



14 16 Flexuous rods from
orange tree (*Citrus
sinensis*) infected with
tristeza virus



14 17 Citrus
tristeza virus on
Kagzi lime (*C
aurantifolia*)
Healthy leaf on
right



14 18 Stem pitting
symptoms of tristeza virus
on *C. aurantifolia*



14.19 Fig mosaic



14.20 Mulberry mosaic

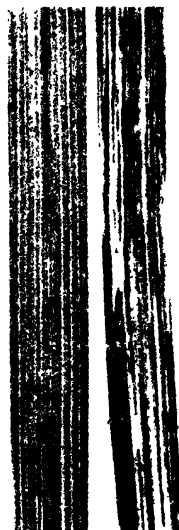
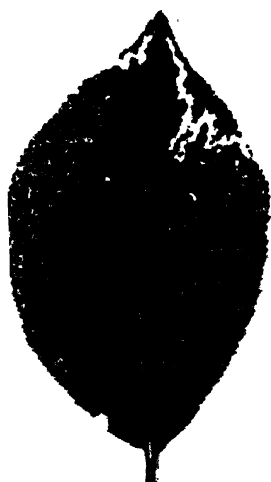


14 21 Mulberry yellow net vein



14 22 Papaya leaf curl

14.23 Plum line pattern



16.1 Ragi mosaic
Left : Healthy leaf
Right : Diseased leaf



1. Electron micrograph of Ragi mosaic virus



16.3 Bajra mosaic



16.4 Bajra streak virus on wheat



16.5 Bajra virus on barley



17 1 Groundnut mosaic



17 2 Groundnut ring spot



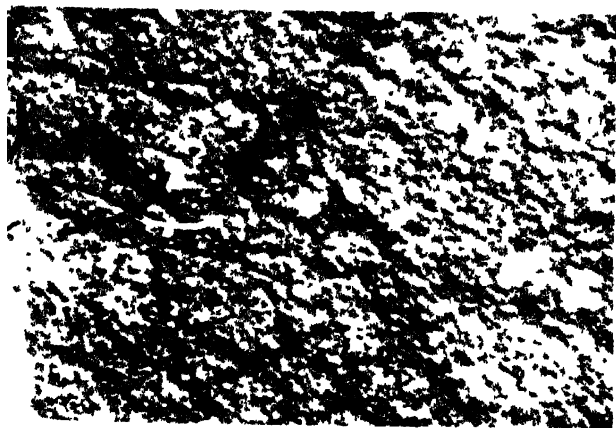
17 3 Groundnut bunchy top



17 4 Groundnut chlorosis



18 1 Canna mottle



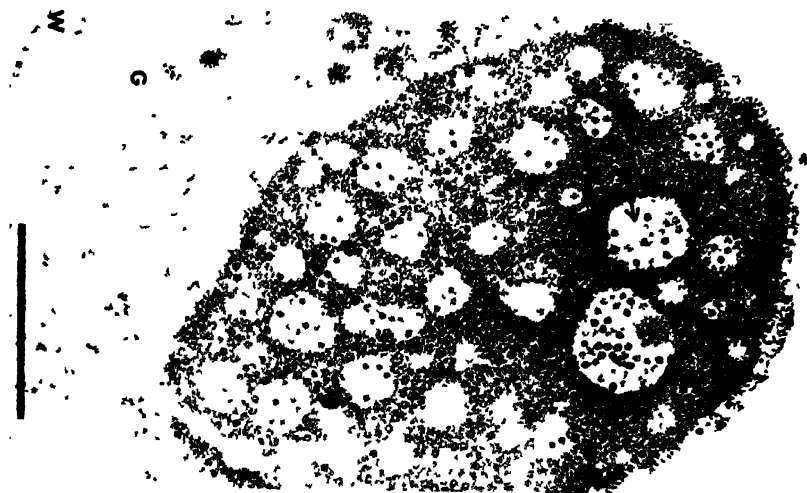
18 2 Electron
micrograph of
Canna mottle virus



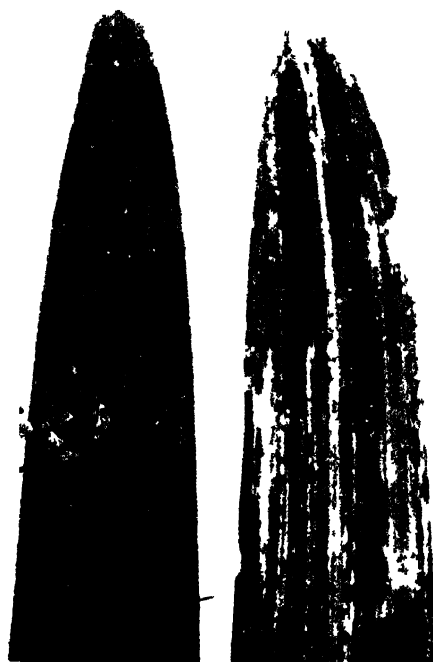
18.3 Dahlia mosaic virus
 Right: Infected Dahlia
 Left: Uninoculated control



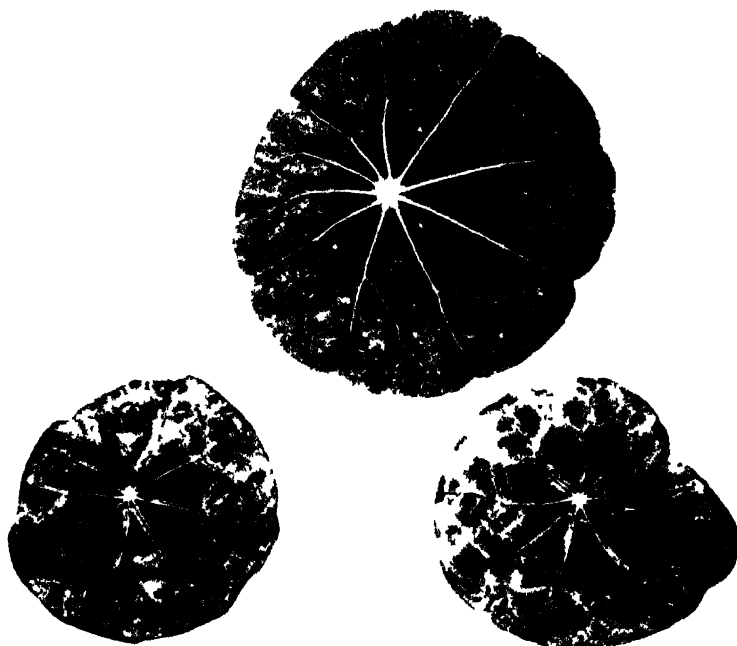
18.4 Dahlia mosaic virus on
 dahlia showing chlorosis along
 the veins



185 Section of leaf of Zinnia infected with Dahlia mosaic virus X 40 000
 V Virus particles G Golgi complex W - Cell wall



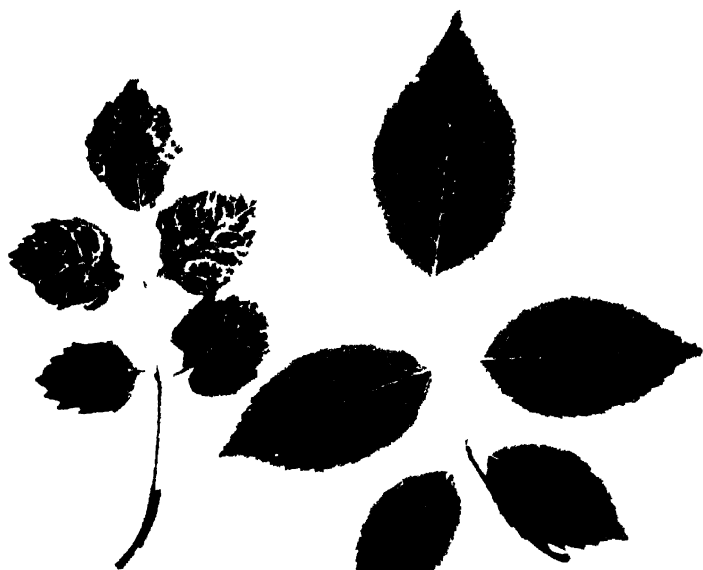
186 Hippeastrum leaves
 affected with mosaic disease
 Left Healthy leaf
 Right Diseased leaf



187 Mosaic of *Tropaeolum majus*



188 Mosaic of *Vinca rosea*



18 9 Yellow vein mosaic of *Rosa bourbina*



18 10 *Hibiscus rosa-sinensis* leaf curl



19 1 Cassava mosa



19 2 Coconut root
(wilt)



19 3 Electron micrograph of
coconut (root) wilt virus
X 30 000



19 4 Mosaic of sugarcane



19.5 Long flexuous rod shaped particles of sugarcane mosaic virus X 60,000



19.6 Tea rose yellow mosaic



19.7 Tea phloem necrosis



19.8 Tobacco
broken ring spot



19.9 Tobacco etch virus



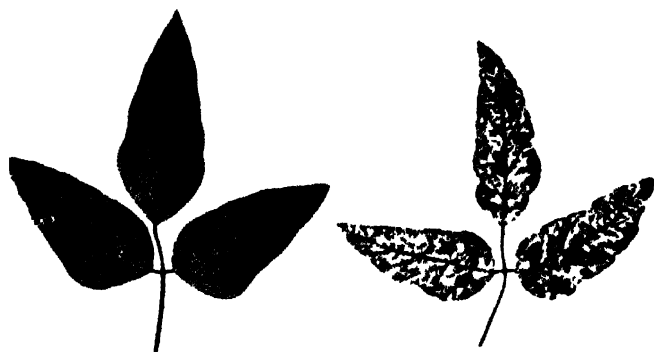
20.1 Healthy and sterility
affected pigeon pea plants
Left Healthy plant bearing
flowers
Right Diseased



20.2 Pigeon pea sterility mosaic
Left Healthy leaf
Right Diseased leaf



20.3 Yellow mosaic of mung
(*Phaseolus aureus*)



20 4 Soybean
mosaic



20 5 *Aceria cajani*
the eriophyid mite
vector of pigeon
pea sterility virus



21 1 Kettle disease
of small cardamom
(a) Healthy leaf
(b) and (c)
Diseased leaves



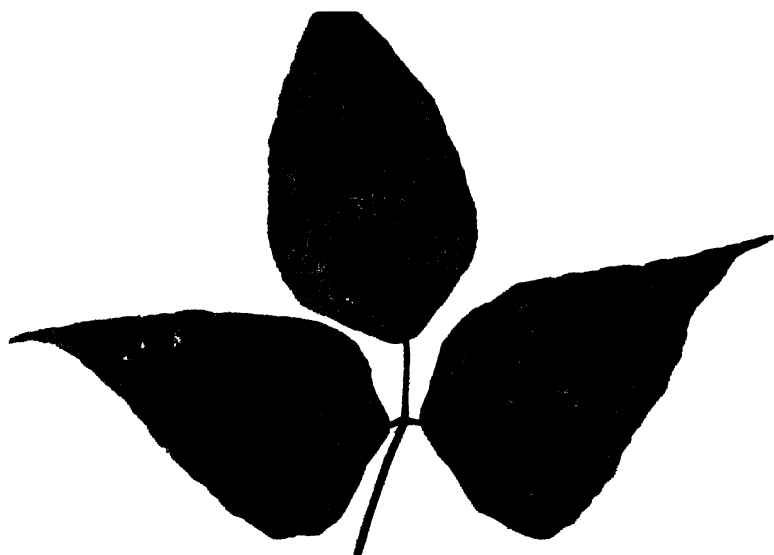
21.2 Chirke disease
of large cardamom



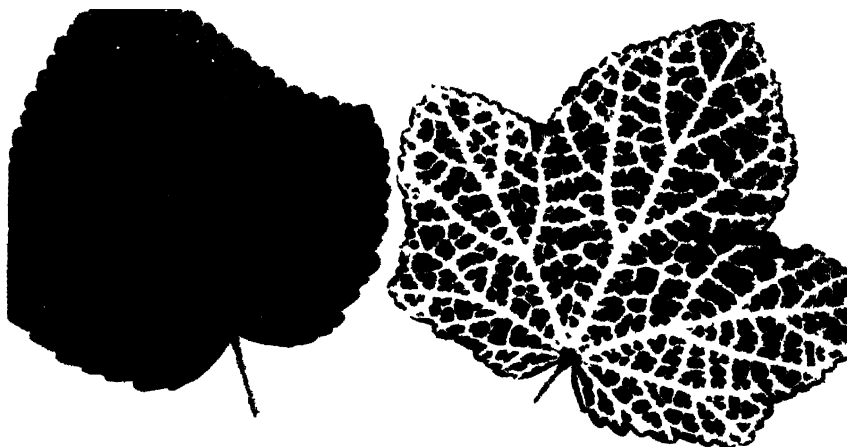
21.3 Large cardamom affected by Foorkey virus
(a) Foorkey infected plant (b) Healthy plant



22 1 Beet root purple top



22 2 Mosaic of bean (*Phaseolus vulgaris*)



22.3 Yellow vein mosaic of *Bhindi* (Okra)

Left Healthy leaf

Right Diseased leaf



22.4 Mosaic of chili



22.5 Cucumber mosaic (*Cucumis* spp.)

22 6 Snakegourd mosaic (*Cucur*
virus 1)



22 7 Electron micrograph of CMV
(*Cucumis* virus 1)



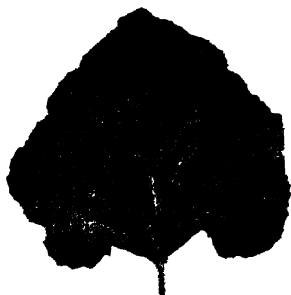
22 8 Bottlegourd mosaic (*Cucumis* virus 2)



22 9 Watermelon mosaic (*Cucumis*
virus 2)



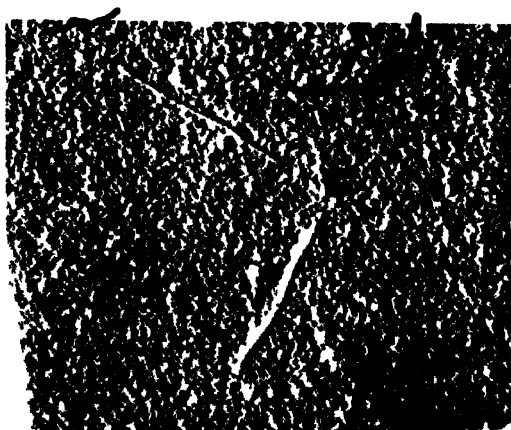
22 10 Electron micro-
graph of (*Cucumis*
virus 2)



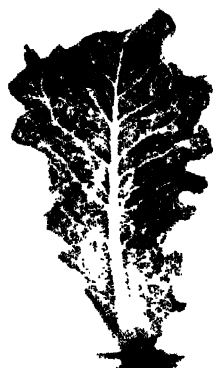
22 11 Pumpkin mosaic (*Cucumis virus 3*)



22 12 Mosaic of *Cucurbita pepo* (*Cucumis virus 3*)



22 13 Electron micrograph
of (*Cucumis virus 3*)



22.14 Lettuce mosaic



22.15 Mosaic of garlic



22.16 Potato mosaic



22 17 Leaf roll of potato



22 18 Veinal necrosis induced
by potato virus Y in potato



22 19 Enation leaf curl of tomato



24 1 Apple rubbery wood



24 2 Greening affected mandarin tree



24 3 Grape fruit affected with greening showing defoliation of leaves



24 4 A leaf of greening affected sweet orange showing yellowing of midrib and lateral veins



24 5 Greening affected kagzi lime



24 6 *Diaphorina citri*
Kuway the vector of
citrus greening



24 7 Fried egg shaped
colony of citrus greening
mycoplasma on PPLO
agar medium



24.8 Small leaf disease of cotton
 Left : Healthy
 Right : Diseased



24.9 Mycoplasma-like bodies in
 the phloem sieve cells of small-leaf
 affected cotton leaf



24 11 Marginal Flavesence
of potato



24 12 Witches broom
disease of potato



24.13 Mycoplasma-like bodies in the potato witches' broom affected cells



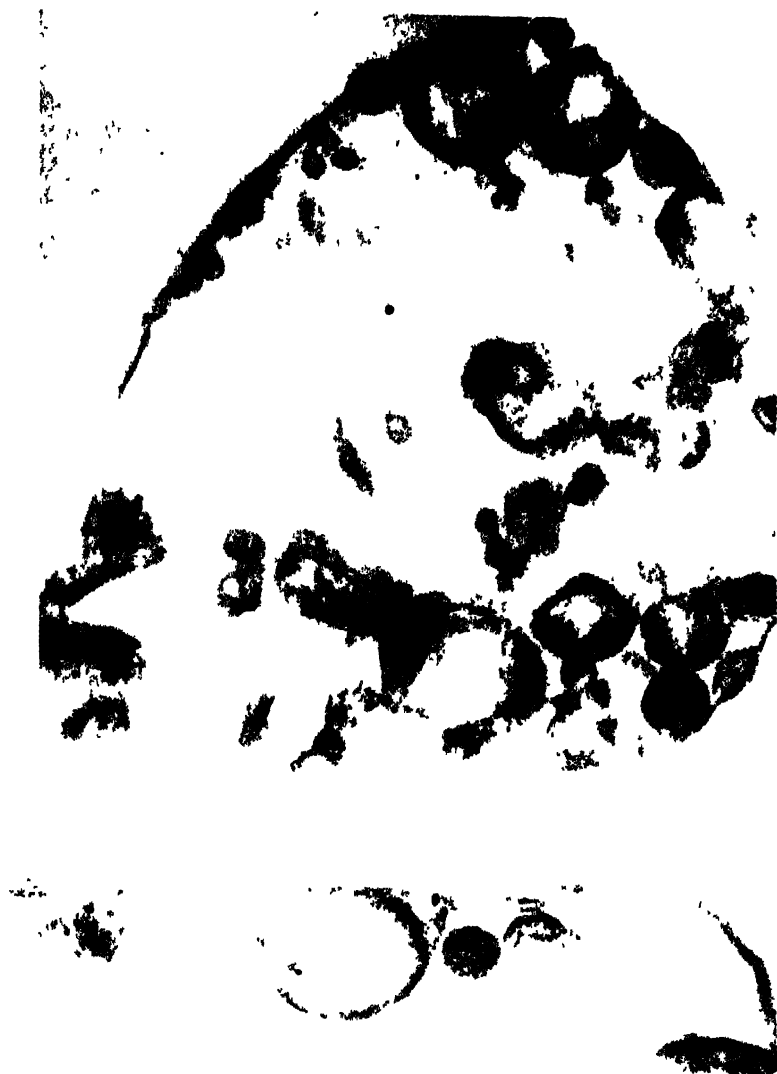
- 24 15 A Spiked sandal tree
 B Healthy sandal tree
 C MLB in plant cells under electron microscope
 D Little leaf affected brinjal plant



24 16 Diseased sandal shoot after treatment
 Note—appearance of healthy leaves



24.17 Sugarcane grassy shoot



24 18 Mycoplasma-like bodies in the sieve tubes of sugarcane affected with grassy shoot



24 19 Sesamum affected with phyllody



24 20 Mycoplasma-like bodies (M) in phloem cells of Sesamum affected with phyllody



6.1 Detection of citrus greening by fluorescent antibody technique. Stained mycoplasma like-bodies in phloem cells show apple green fluorescence

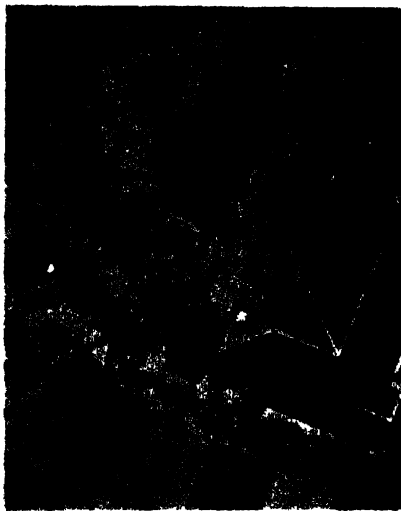
6.2 Detection of Cowpea mosaic virus in cowpea leaves by fluorescent microscopy. Epidermal peelings show virus inclusions adjacent to the nucleus



6.3 (a) Detection of Cowpea mosaic virus in seed by fluorescent microscopy. Cells from diseased seed showing intense red fluorescence



6 3 (b) Detection of Cowpea mosaic virus in seed by fluorescent microscopy. Cells from healthy seed



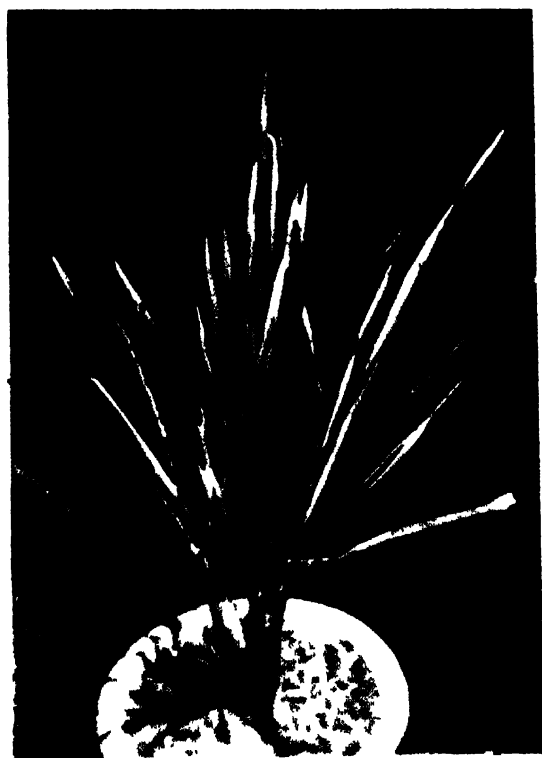
6 4 Detection of coconut root (wilt) by aerial infra red photography. Trees in circles show severe disease intensity



10.1 Maize mosaic



106 Bajra streak



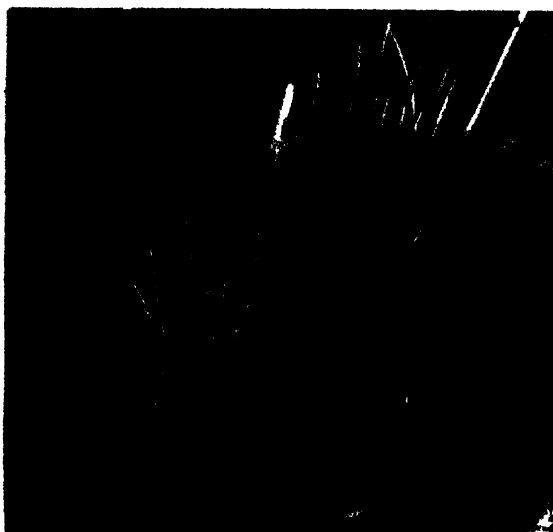
Rice tungro virus



0 8 Mosaic streak of wheat



24 10 Peanut witches broom



24.14 Rice yellow dwarf

Plantation Crops

ARECA CATECHU

Yellow Leaf

Symptoms: The disease is characterised by the appearance of translucent spots, 1 to 2 mm in diameter in the growing spindle of the palm. Brown necrotic streaks, running parallel to the lamina, are present in the unfolding leaves. As the leaf develops, yellowing starts from the tips of the leaflets, gradually extending to the entire lamina; there is an abrupt demarcation of the yellow and green areas of affected leaves. In advance stages of the disease, the leaves are reduced in size, are short, stiff and pointed, closely bunched and abnormally puckered. Ultimately the leaves fall off, leaving a bare pointed trunk. At this condition, the stem becomes friable, conducting strands break off in masses and black discolourations are sometimes present. The root system is also affected, the tips of young roots are dark and gradually rot. The fruits become black and fall off in large numbers.

Transmission: The virus is mechanically transmissible. It is also soil-borne but the vector is not known.

Host range: Includes *Hibiscus esculentus*, *Manihot utilissima*, *Cocoa nucifers* and *Vigna* sp (Menon, 1963). The disease is found to occur in Southern India, i.e., in all parts of Kerala, Karnataka, Tamil Nadu and Maharashtra (coastal regions).

CACAO

Swollen Shoot

The swollen shoot disease of cacao in West Africa is caused by a

complex of viruses some of which are closely related strains and are given below:

Mottle leaf

Symptoms: Mottling of leaves is the chief symptom exhibited by the plants. Clearing and vein banding of veinlets between the main veins, but not alongside veins are also observed. No swellings have been observed on infected plants. Pod size may reduce considerably (Posnette, 1947; Thresh and Tinsley, 1959).

Transmission: The virus is transmitted by the insect vector, *Pseudococcus citri*. Kenten and Legg (1967) have reported transmission by seven species excluding *Ferisiana virgata* Ckll.

Properties: Expressed sap is not infective but infective extracts can be prepared from the leaves using antioxidants. Such extracts infected upto 3 per cent of inoculated cacao beans (Brunt and Kenten, 1962). The thermal inactivation point ranges between 50 to 60°C for ten minutes, the dilution end-point is 1:100 and longevity *in vitro* is ninety-six hours at 0° to 25°C. The virus remains still infective after freezing *in vitro* and is precipitated from clarified extracts by the addition 150–200 g/l ammonium sulphate. The particles are rod shaped 143 × 25–26 nm with rounded ends (Kenten and Legg, 1967).

Host range: The virus infects *Abroma augusta*, *Sterculia rhinopetal*, *Theobroma bicokr*, *T. obovatum*, *T. speciosa*, *Adansonia digitata*, *Bombax buonopozense*, *P. beau*, *Ceibra pentandra*, *Cercorus aestuans*, *C. tridens* (Legg and Bonney, 1967).

The disease occurs in Ghana and Nigeria.

Symptoms: The virus causes Quite necrotic infection followed by recovery phase with limited leaf symptoms, but no swellings and no red vein-banding or mottle on leaves. Translucent distorted patches appear on the hardened leaves (Kenten and Owusd, 1970).

Transmission: The virus is mechanically transmissible. It is not transmitted by mealy bugs. Particles are isometric, 24–26 mm in size.

Host range: The virus infected 11 of 25 harbaccons hosts tried including *Phaseolous vulgaris* var. Prince and *Beta vulgaris*.

Swollen Shoot Virus Strain A

Symptoms: The disease is characterised by the swelling produced on the branches and on the top and lateral roots. Swellings are

sometimes pronounced on suckers arising from the base of the trunk, amounting to twice the diameter of the normal stem. On fan branches the swellings are slight. Vein clearing, vein banding and opaque yellowish green areas develop on the leaves.

In the young leaf soon after emergence from the bud, the veins are bordered by a narrow red band. In the beginning of infection this forms a fine red network over the lamina. Pods of infected plants are smaller and contain only about half the weight of beans of a normal pod, the beans are flatter, often containing cotyledons paler than usual (Posnette, 1947).

Transmission The virus could be transmitted mechanically by sap partially purified by ammonium sulphate precipitation by differential centrifugation (Kenten and Legg, 1970). The insect vectors of the virus are *Pseudococcus njalensis*, *P. citri*, and *Ferrisia virgata* (Posnette and Strickland, 1948).

Strain B

Symptoms Swellings are produced on branches and roots as with strain A, but are more pronounced and usually more elongated. The network of fine veins on leaves is cleared and banded with yellow. No pod symptoms were noticed. Affected plants are not stunted.

Transmission The insect vectors are *Pseudococcus njalensis* and *Ferrisia virgata* (Posnette and Strickland, 1948).

Strain D

Symptoms are usually pin-point yellow spots, evenly distributed over the leaf. Swellings are seldom formed but occur occasionally as slight internodal inflations near the tips of branches. Vein clearing occurs rarely. In later leaf flushes, symptom bearing leaves are few and show a distinctive mosaic of 'pepper and salt' type, composed of both fine yellow dots and larger cleared flecks.

Transmission The insect vectors are *Pseudococcus citri* and *P. njalensis* (Posnette and Strickland, 1948). All the above viruses are common in Nigeria and Ghana.

Control

Eradication: Infected trees are removed only from the surrounding area (Thresh, 1959).

Vectors. Control of mealybugs with parasitic fungi although promising in laboratory tests, likewise proved unsuccessful in field trials (Nicol *et al.*, 1950).

Protection of mild strains: Infecting trees with mild strains to protect them against the effects of severe strains was earlier considered to be useful in special circumstances (Posnette and Todd, 1951, 1955; Thresh, 1958).

Use of resistant and tolerant varieties: As other methods of control are either ineffective or too costly, the production of tolerant or resistant cacao varieties seems to be the best way to control swollen shoot disease (Longworth and Thresh 1963; Legg and Kenten, 1968, 1971a, b; Kenten and Legg, 1971b).

The viruses infecting cacao in Ceylon cause leaf symptoms resembling those caused by some strains of cacao swollen shoot virus (Peiris, 1953) and are transmitted by mealybugs (Carter, 1956). Orellana and Peiris (1957) found nodal and internodal swellings on 300 trees, it seems that several strains of a virus resembling cacao swollen shoot virus infect cacao in Ceylon.

Symptoms resembling those caused by viruses have been noted in cacao in Venezuela (Posnette and Palma, 1944), Java (Thung cited by Thresh, 1958; Semangun, 1961), Sabah (Reddy, 1968) and Costa Rica (Hutchins, 1959).

In Trinidad two other viruses or virus strain have been observed to be different from those of swollen shoot complex of W. Africa. They were first reported by Posnette (1944) who named them as 'red mottle' and 'vein clearing' diseases. Later Baker and Dale (1947) named these as strain A and strain B.

Strain A or Red Mottle

Symptoms: The virus causes a red pigmentation of the tissues bordering the main veins of the leaf. Red mottle is usually accompanied by the same form of mosaic and develops cleared areas, varying in size and shape. Red blotches on the young pods of certain yellow podded varieties may develop and die back and reduction in yield occur in fairly young trees.

Transmission: The virus is transmitted by budding only.

Strain B or Vein Clearing

Symptoms: Chief symptoms other than strain A are the development of a pronounced vein clearing which may extend to the fine veins of the leaf and persists after hardening of the leaves.

Transmission: The virus is transmitted by budding only.

Mosaic

Four mosaic patterns have been met with, on cacao leaves from Java, namely, vein clearing, oak leaf pattern, and scattered chlorotic spots, all similar to those caused by viruses in W. Africa and Ceylon. Stem and root swelling and reduction of growth have not been observed (Semangun, 1961).

Further information is lacking.

Yellow Mosaic

Symptoms: The disease is characterised by the development of large circular chlorotic blotches which often become completely chlorotic and take the form of yellow mosaic (Blencowe *et al.*, 1963). The virus has been reported from Sierra Leone.

Transmission: The virus is easily transmitted by mechanical means. Vector is not known yet.

Properties: The thermal inactivation point ranges between 60-65°C with ten minutes exposures, the longevity *in vitro* is between sixteen to thirty-two days at 25 to 30°C. The dilution end-point is 10^4 .

The particle measures about 25 nm in diameter. The particle contain single stranded RNA. The virus is serologically related to turnip yellow mosaic and wild cucumber mosaic virus (Brunt *et al.*, 1965).

Host range: The virus infects *Vinca rosea*, *Begonia*, *Adansonia digitata*, *Ceiba pentandra*, *Pachira*, *O. eleagina*, *Beta vulgaris*, *Chenopodium amaranticolor*, *C. quinoa*, *Cucurbita facifolia*, *Malox pepo*, *C. pepo*, *Cucumis melo*, *C. sativus*, *Luffa cylindrica*, *Momordica charantia*, *Nicotiana tobacum*, *N. glauca*.

CASSAVA (*Manihot utilisima*)

Mosaic

Symptoms: The disease is characterised by chlorosis of the leaves in the form of white to pale yellow areas alternating with green ones. The leaves are also reduced in size, malformed and twisted (Raychaudhuri, 1967). Great variations occur, however in the symptom manifestation and, Storey and Nichols (1938a) have analysed into three steps (a) chlorotic, (b) size of the chlorotic areas (c) frequency of the chlorotic areas, etc. The disease has been reported from Brazil, Africa, New Guinea and India.

Transmission: Leferve (1945) reported that the virus is transmitted by mechanical inoculation. But all attempts by Storey and Nichols (1938b) failed. The disease is transmitted by grafting but not by sap inoculation. Incidence varies from 60 to 100 per cent (Alagianagalingam and Ramakrishnan, 1966). The virus is not carried through true seed. However, it is known that in nature whitefly *Bemisia* species is responsible for disease spread. The insect can transmit the virus only through the immature leaves (Raychaudhuri, 1967; Alagianagalingam and Ramakrishnan, 1966).

Properties: Virus particles measuring 10-45 nm in diameter, loosely associated into fibrous masses were detected in nearly all infected tissues except tracheids and sieve tubes. The virus particles were not associated with any particular cell structure and the cells containing these showed significant accumulation of starch in the plastids (Kitajima and Costa, 1966).

Host range: Cucumber is a herbaceous host of the cassava mosaic virus (Menon and Raychaudhuri, 1970). Field testing of cassava selections and hybrids have indicated that S-1310, S 1315, S-237, S-2380, H-97, H-43, H-86, H-165 and H-226 are resistant (Chacko and Thankappan, 1969).

Resistant varieties tend to remain symptom free. Symptoms when occur are usually mild frequently restricted to the roots. In hybrids symptoms are either severe or absent (Jennings, 1960).

Control: Use of healthy cuttings. Grow resistant varieties.

Stem Lesion

Symptoms: The disease manifests as dark brown stripes on the otherwise green stems and yellow mottling of the leaves. The stem lesions remain as sunken areas when the bark of the stem peels off. If badly infected the diseased stem becomes brittle and readily breaks off at ground level. The leaf mottling is of a different character from that in mosaic that is, only leaf mottling occurs and no mosaic pattern is formed along with it (Storey, 1936). The disease has been reported from Africa.

Transmission: The virus is transmitted by grafting only. No other information is available.

Control: The control of the disease is not yet known.

COCONUT

Root Wilt

Symptoms: The main symptoms of disease are wilt accompanied by flaccidity of leaflets and abnormal bending of the leaves. The inner whorls of leaves often show yellowing. The leaves are stunted and the crown is reduced. The disease is very widespread in Kerala, India.

Transmission: The virus is transmitted by mechanical inoculation and also by Tingid bug *Stephanitis typicus* (Nagraj and Menon, 1956). The virus is also soil-borne and could be isolated from the soil taken from the base of the diseased trees and the recent evidence indicates that the virus is also present in coconut milk.

Host range: Cowpea (*Vigna sinensis*) has been in use as an indicator host. It produces abnormalities of trifoliate leaves due to infection with the pathogen (Shanta and Menon, 1960). The reaction is however, erratic and seems to be very sensitive to climatic factors. The insect vector *S. typicus*, gives better percentage of infection in cowpea seedlings although it is forced to feed on it. A minimum of viruliferous insect per seedling can produce 5 per cent infection in cowpea seedlings (Nagraj and Menon, 1956). The purified virus preparation is infective to tobacco and induces local lesions on *Chenopodium amaranticolor*. Summanwar *et al.* (1969) purified the virus from samples obtained from diseased roots and leaves and with the help of electron microscopy established the association of rod-shaped virus particles measuring 320-360 \times 24-25 nm. The virus is serologically related to tobacco mosaic virus (Summanwar *et al.*, 1971).

Properties: The virus is inactivated by exposure at 90°C. The dilution end-point ranges between 10^{-6} to $10^{-6.5}$. Longevity *in vitro* is about a year.

Control: No control measures except judicious manuring and cultural practices are recommended. Eradication of disease in new areas is under experimentation. No resistance has yet been located in indigenous and exotic varieties of coconut.

Cadang Cadang

Symptoms: The characteristic symptoms of the disease are water soaked spots without brown centre and are clearly demonstrated from the lamina, occasionally aggregating into streaks parallel

to veins. All nut bearing trees with such spots possess an additional diagnostic feature the roundish, and scarified nuts (Nagraj *et al.*, 1965). According to Price (1971) the disease symptom are a rounding of the nuts which became restricted with dark scarifications and spots appear as bright yellow. The disease has been reported from Philippines, India and Guam.

Transmission: The disease is transmitted by *Aphis gossypii*, colonising naturally on *Borreria articularis* and uris-foca aphids reared in the laboratory on *Commelina benghalensis*.

The disease is also transmitted through needle pricks (Celino, 1947). Reinking and Radewald (1961) consider that the disease may be caused by a soil-borne plant virus spread by dagger nematodes.

No bacteria, fungus, alga or nematode has been consistently associated with the disease. (Price, 1971).

COFFEE

Blister Spot

Symptoms: Symptoms consist of yellowish round convex and blister-like spots on young leaves beaming larger and coalescing in the older leaves. Growth is stunted, internodes shortened, flowers few in number and setting very few fruits which are generally smaller than in healthy plants. Many of these turn black towards the terminal half of fruit branches and develop round. Slightly sunken lesions (Wellman, 1957). The disease has been reported from Costa Rica.

Transmission: The virus is graft transmissible only (Reyes, 1959).

Ring Spot

Symptoms: The disease is characterised by the appearance of circular translucent ring spots from pinhead size to 1.5 cm in diameter or concentric or complete necrotic rings and line pattern on the leaves (Bitancourt, 1958).

The disease has been reported from Brazil.

Transmission: Natural spread is very slow, infection is apparently directly from tree to tree.

SUGARCANE

Fiji Disease

Symptoms: The disease is characterised by the development of elongated swellings or galls on the under surface of the leaves. These galls extend along the larger veins or vascular bundles and are in fact, formed by the abnormal growth of the tissues comprising the vascular bundles. Galls are produced in similar manner in the vascular bundles of the stem and may be detected by splitting open the affected shoot.

In field a diseased shoot may attain a fair length and be clothed with many healthy looking leaves of the usual length and colour, but suddenly it loses power to produce normal leaves, throws out a few bent and twisted stumps and then ceases to grow altogether. The shoot may remain alive for months or it may soon die. When such shoot is examined the characteristic galls are usually to be found on most of the healthy looking leaves which are not otherwise distorted and on all of the deformed aborted leaves.

The disease is cumulative in the cane: the galls mark well advanced stage of the disease and the distortion of the apical leaves is its final culmination (Lyon, 1921).

Transmission: The virus is not mechanically transmitted. There is no evidence of transmission through seed or soil. *Purkinella vastatrix* is known to transmit the disease in Philippines (Octemia, 1934).

Host range: Fiji disease virus seems to be confined in its host range to sugarcane. The disease has been reported from New South Wales, Java, Philippines and New Guinea.

Control: By the use of more resistant varieties.

Mosaic

Symptoms: The chief symptoms of the disease are the appearance of pale patches or blotches in the green tissues of the leaves. These are generally irregularly oval or oblong in outline, their longer axis parallel to the midrib. They are not confined between veins, and consequently are not of uniform width throughout any considerable part of their length.

Transmission: The virus is transmitted mechanically. It is seed-borne in small percentage. The vectors are numerous aphid species (Tate and Vanderberg, 1939) as *Rhopalosiphum maidis*,

Carolinaia cyperi, *Aphis gossypii*, *Schizaphis graminum* and *Hysteroneura* (Chona and Seth, 1958; Seth and Chona, 1961).

Properties: The thermal inactivation point is 53 to 55°C, dilution end-point is 1:1000 (Adsuar, 1950) and longevity *in vitro* is two to twenty-four hours (Rafay, 1935, 1957). The virus particles measure 760 ± 10 nm in length and 12 nm in width (Herold and Weibel, 1963).

Host range: The virus infects maize, sorghum, pearl millet, Sudan grass, tueros grass, wonder forage grass and other wild grasses.

Control: The use of resistant varieties of sugarcane, systematic roguing of infected canes, the use of selected healthy setts and legislation.

The disease occurs in all the sugarcane growing countries of the world. In India, sugarcane mosaic was first noticed at Pusa in 1921. It is now widespread in all cane growing areas.

Ratoon Stunting

No clear symptoms of this disease usually appear on the affected plants except that such plants keep on declining in vigour and yield with each vegetative generation, in some varieties. The fibrovascular bundles in the nodal region become reddish and can be observed if the cane stalk is split open (Singh, 1966).

The disease has been observed in Queensland, New Guinea and India (Rafay, 1957).

Transmission: The disease is transmitted through harvesting implements. The disease is readily transmitted to healthy plants by mechanical inoculation. Primary infection arising from the planting of diseased setts appear to be the most important method of spread of the disease (Singh and Rao, 1969).

Properties: The thermal inactivation point is 55°C and the longevity *in vitro* is two days (Farrar, 1957).

Host range: Host range includes paragrass (*Brachiaria mutica*) *Cynodon dactylon* and Guinea grass (*Panicum maximum*) which are symptomless carriers (Steindl, 1957).

Control: The disease is controlled effectively by hot air treatment at 54°C for eight hours and hot water treatment at 50°C for two hours respectively (Singh, 1966; Srinivasan and Rao, 1968). Treatment with hot air at 129°F (54°C,) for eight hours has been found to eliminate the virus without causing such heavy reductions in

germination (Launden, 1953; Hughes, 1953; Knust, 1953).

Streak

Symptoms: The disease resembles mosaic except for the fact that the yellowish spots have well defined border (France, 1956).

Transmission: The virus is transmitted by *Tomasis liburata* (France, 1956; McClean, 1947). The disease has been reported from Brazil.

TEA ROSE (*Camellia japonica*)

Yellow Mosaic

Symptoms: The affected leaves show pronounced yellowing of leaf tissue along the veins. The dark green vein banding along the yellow areas is conspicuous (Sharma and Raychaudhuri, 1972).

Transmission: The virus is not sap transmissible and can be transmitted by grafting only. *Toxoptera aurantii* and *Cuscuta reflexa* transmitted the virus (Ahlawat and Sardar, 1973).

Host range: Apart from *Camellia japonica* the virus infects tea, *Camellia sinensis*. It occurs in nature on tea in many tea estates.

Control: Control of the disease is not yet known. The disease has been reported from India.

CAMELLIA THEA

Phloem Necrosis

Symptoms: The necrosis in the root appears to be the most constant single diagnostic feature of the disease at the early stage of infection. The necrosis is identified by falling of a slice of bark of the root so that the phloem is exposed close to the cambium. Later the phloem necrosis has been found to occur in the aerial parts of the plant. In the aerial shoots symptoms take the form of a backward arching in mild cases often symptoms take the form of a inward folding of the lamina along the mid rib and in severe cases resulting in twisting and crumpling of the apical portion of the leaf. The severe curl is usually associated with a zigzag habit of growth (Bond, 1944a, b). Bond (1949) has reported the methods of differentiating between the true phloem necrosis of the disease and false necrosis of the non-pathological origin. These two may have some logical affects except the latter has no

hyperplasia in the petiole, difference can be identified on the basis of their position as seen in a transverse section of the bundle.

Transmission: The virus is transmitted only by various types of grafting including seedlings grafts and root grafts. No insect vector is known.

Host range: The virus is restricted to tea plant only and has been reported from Ceylon.

TOBACCO (*Nicotiana tabacum*)

Broken Ring Spot

Symptoms: The disease is characterised by development of isolated rings or ring and line patterns on the developing leaves of tobacco plants. As these rings possess incomplete wall that is non-necrotic areas alternating with the necrotic patches the virus has been named as the tobacco broken ring (Sastry, 1965). The disease has been observed in India.

Transmission: The virus is mechanically sap transmissible. The insects, namely, *Myzus persicae*, *Aphis gossypii*, *A. evonymi*, *A. craccivora*, *Lipaphis erysimi*, *Bemisia tabaci* and a number of unidentified insects including different species of leaf hopper, collected from field failed to transmit the disease.

Properties: The virus withstood exposure to 55°C but not 60°C at room temperature. The dilution end-point ranges between 1:100 and 1:1000. The longevity *in vitro* at room temperature (35–37°C) is ten days.

Host range: Out of thirty-five species of plants belonging to eleven natural families and twenty-two genera, five showed systemic symptoms, only one localised reaction and none of them is found to be the symptomless carrier.

Distortion

Symptoms: The disease manifests itself as vein clearing on the young leaves followed by severe mottle vein banding, deep green blisters and malformation and downward curling of the leaves are also seen (Azad and Sehgal, 1958). The virus occurs as complex along with tobacco mosaic virus, and has been observed in India.

Transmission: The virus is sap transmissible.

Properties: The thermal inactivation point ranges between 72 to 78°C. The longevity *in vitro* is about nine days at room

temperature. The dilution end-point ranges between 1:150 and 1:180.

Host range: Host range is restricted to solanaceae. The virus causes severe symptoms in tobacco var. *N. glutinosa* and is transmissible to seven other *Nicotiana* species but not to *N. glauva*, potato and *Capsicum* sp., *Datura metel* and *Nierembergia frutescence* are symptomless carriers (Azad and Sehgal, 1958).

Etch Virus

Symptoms: The most characteristic symptom of the disease is definite necrotic rings or faint, diffuse, chlorotic spots on the leaves. In the beginning the disease develops at the base of the leaves and appears as a clearing of the veins with a fine necrotic etching. Vein clearings and etching persist for a few days and are then replaced by general chlorosis. The internodes are shorter than normal thus the whole plant appears stunted.

Transmission: *Aphis craccivora* transmits the virus in a persistent manner (Kassanis, 1941; Herold, 1970). The virus is mechanically transmissible but is not seed borne (Bennett, 1944). The virus has been reported from India in brinjal by Varma and Lal (1964) and a serologically related virus has been shown to cause mild mottle symptoms on tobacco (Verma, 1964).

Properties: The thermal inactivation point ranges between 54-58°C. The dilution end-point of the virus ranges between 1:1000 to 1:5000. Longevity *in vitro* is eight days. Bawden and Kassanis (1941) assume that it is a nucleoprotein with asymmetrical, probably rod-shaped particles. Under electron microscope infected tobacco plants revealed elongated particles. The average length of the 70 per cent particles is 754 nm (Herold, 1970).

Host range: The virus infects tobacco, sweet and hot pepper (*Capsicum frutescence* var. *grossum* and *crasifirma*) tobacco var. White Burley. Also includes *Datura stramonium* tomato, petunia and potato (Holmes, 1946).

The disease has been found in Germany, Japan, Canada, USA and South America.

Symptoms: The diseased is characterised by dark green mosaic and with occasional necrotic spots on the leaves (Verma, 1964).

Transmission: The virus is sap inoculable.

Properties: Thermal inactivation point ranges between 50-60°C. The virus retains infectivity for fifty-six hours at room temperature

(28 to 30°C) and for ten days at 0°C. The dilution end-point ranges between 1:100 to 1:1000. Serologically the virus was found to be a strain of tobacco etch virus (Varma and Lal, 1964). **Host range:** The virus infects different varieties of *N. tabacum*, *N. glutinosa*, *N. trigonophylla*, *N. rustica* var. C-302 and NPS 219, two species of *Datura* (*D. stramonium* and *D. metel*).

Leaf Curl

Symptoms: The chief symptoms of the disease are the curling of the leaves, clearing and thickening of the veins, twisting of petioles in severe cases and the presence of enations or leafy out-growths on the veins on the under surface of the leaves. The leaves are puckered, rugose, brittle and reduced in size to varying degrees depending on the severity of the strain. The internodes are shortened resulting in dwarfing of the plants.

Transmission: The virus is transmitted by grafting and through an insect vector, white fly *Bemisia tabaci*.

Pruthi and Samuel (1937, 1939, 1941) studied the virus-vector relationship of the virus and alternate host plants of the virus. **Host range:** The virus infects tomato, sunn hemp, zinnia, petunia, chilli, datura, papaya, *Physalis peruviana*, *Schizanthus* sp., *Ageratum conyzoides*, *Solanum nigrum*, *Euphorbia turtis*, *Veronia*, *Caneris*, *Launes asplenifolia*, *Sida rhombifolia*, *Scoparia dulcis*, and *Withania somnifera*.

Control: The disease can be controlled by eradication of diseased plants and complete destruction of ratoon tobacco and slumps of the previous crop.

The disease is prevalent in Belgium, Congo, India, Java, Madagaskar, Nyasaland, Rhodesia, South Africa, Tanzania, Venezuela and Zanzibar.

Mosaic

Mosaic disease is prevalent in all places wherever tobacco is grown. It is the most widespread and highly infectious.

Symptoms: Infected plants show a distinct mosaic mottling, distortion of leaves, stunting and retardation of growth. Occasionally leaves develop large blisters of green tissue. Raised or sunken and discoloured areas may also develop.

Transmission: The virus is readily transmitted by sap inoculation and through contact in field. It is believed that the virus is

transmitted through seeds of tomato to a very limited extent. So far there is no convincing evidence of insect transmission of the virus (Smith, 1957). However, it is shown (Walters, 1952) that some grass hoppers (*Melanoplus* sp.) could transmit the virus by mechanical contamination of the mouth parts.

Properties: The virus is inactivated by exposure to 93°C for ten minutes but can withstand lower temperatures for longer period. The tolerance to dilution exhibited by the virus is 1:1,000,000. Longevity *in vitro* is several years. In dried state and in desiccated leaf tissue the virus retains infectivity for a year or more. The virus particles are rod shaped and measure approximately 280 nm long by 18 nm in diameter.

Host range: Host range of the virus is very wide. Several *Nicotiana* spp. such as *N. glauca*, *N. rustica*, *N. sylvestris*, *Datura stramonium*, tomato, brinjal, *Solanum nigrum*, *Physalis peruriana*, Petunia, chilli, spinash, beet, phlox, etc.

Control: Sanitary measures. Infected plants in seed beds should be rouged out and carefully burnt. Removal of solanaceous weeds from the seed beds, to prevent spread by contact. Smoking or chewing of tobacco may be avoided by the workers while working in the fields.

The virus exists in several strains, the important being Holme's Rib-grass strain, tobacco distorting virus (*Nicotiana virus* IA), Ring spot strain and tomato aucuba strian (*Nicotiana virus* 1C).

From India. Nariani and Nyako (1963) reported another tobacco mosaic disease caused by *Cucumis virus* I.

Symptoms: The disease is characterised by general chlorosis of the leaves with occasional dark green blisters. The leaves are leathery to touch and are invariably narrow with painted tips and wavy margins. Affected plants are stunted and bear fewer flowers.

Transmission: The virus is sap transmissible but is not seed borne. *Aphis gossypii* and *Mysus persicae* are the vectors of the virus.

Properties: The thermal inactivation point lies between 50–55°C. The dilution end point lies between 1:100 and 1:250. Longevity *in vitro* is eight to twelve hours at room temperature (17–22°C) and 24–48 hours at 9–10°C.

Host range: Several *Nicotiana* species could be systemically infected with the virus. These include *N. glutinosa*, *N. rustica*, *N. paniculata*, *N. glauva*, *N. sylvestris*. The virus also infects chilli,

cucumber, *Cucumis melo* var. *Utilissima*, *Cucurbita pepo*, *Luffa acutangula* and *Trichosanthes anguina*. It produces local lesions on *Vigna sinensis*, *Beta vulgaris* and *Spinacea oleracea*.

Control: Use of insecticides is advocated to prevent the spread of the disease in the field.

Ring Spot

Symptoms: The disease is characterised by the development of necrotic rings, varying considerably in diameter depending upon the environmental conditions.

Transmission: The virus is transmitted by *Xiphinema americanum* (Fulton, 1962).

Host range: The hosts *Abutilon theophrasti* and *Polygonum hydropipenoides* proved susceptible to tobacco ring spot virus while seven other common weeds were not infected. Attempt to transmit the virus from diseased to healthy soybeans by various arthropod vectors were unsuccessful (Dysort and Chamberlain, 1960).

Yellow Net

Symptoms: The first recognisable symptoms are a few scattered yellow circular spots on the leaf lamina. After the yellow spot phase, the yellow net symptoms start appearing at the tip of the leaves and spread downwards. Later chlorosis extends to the adjacent interveinal areas resulting in large green islands surrounded by yellow tissue. Complete yellowing of the older leaves may result in some cases.

Transmission: The disease is transmitted by grafting only. *Bemisia tabaci* is found to be its insect vector.

Host range: Host range of the virus includes *Nicotiana glutinosa*, *N. glouca*, *N. repanda*, *N. rustica*, *N. megulosphum*, *N. longiflora*, *N. sylvester*, *N. paniculata*, *N. alata*, *N. companiculata*, *N. grandiflora*, *N. debneyii*, *Lycopersicon esculentum*, *Solanum nodiflorum*, *Beta vulgaris* and *Aster* (Dhingra and Nariani, 1962). The disease has been observed in India.

Streak

Symptoms: The disease is characterised by necrotic local lesions surrounded by concentric water soaked lines or rings which later become brown and necrotic. The lesions tend to spread along the veins, parallel necrotic lines appearing in the surrounding tissue

and sometimes causing the collapse of the midrib and petiole. Affected leaves are small, narrow and slightly crinkled. Leaves may be affected on one side only, the midrib curling towards the affected side (Berkeley and Philips, 1943).

Transmission: The disease is transmitted by dodder *Cuscuta campestris* (Fulton, 1948) and also by mechanical means (Diachun and Valleau, 1950).

Properties: The thermal inactivation point is 53°C, the dilution end-point ranges between 1:20 to 1:100 and the longevity *in vitro* is twenty four to thirty-six hours at 22°C.

The particles are isometric and measure 27 to 29 nm and 34 nm (Kitajima, 1968).

Host range: It infects burdock, white clover hedge mustard, bindweed and plantain.

The disease has been found to occur in Brazil.

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20

Pulses

CHICK PEA (*Cicer arietinum*)

Mosaic

Chick peas are infected in the field with four viruses, namely, alfalfa mosaic, bean yellow mosaic, cucumber mosaic and pea leaf roll (PLVR) (Mosahebi, 1968; Danesh and Kaiser, 1969).

Different species of aphids are responsible for their spread; all except PLRV are mechanically transmissible and none is seed borne. They have all been isolated from different hosts (Danesh and Kaiser, 1969).

Further information is not available about this disease.

COWPEA (*Vigna sinensis*)

Mosaic (*Trinidad*)

Dale (1949) reported the occurrence of a mosaic disease of *Vigna unguiculata* from Trinidad.

Symptoms: The disease is characterised by the appearance of dark and light green rings on the leaves. Later leaves develop irregular yellowish and dark green mottling, accompanied by blistering of the lamina. The final mosaic pattern is rather variable: rough, irregular mottling may be produced but the more chlorotic colouration is usually associated with the veins. Sometimes under glasshouse conditions a reddish brown necrosis of the veins also develops (Dale, 1949).

Transmission: The virus is mechanically transmissible and is seed-borne. The insect-vector is the leaf beetle *Ceratoma ruficornis* in Trinidad (Sale, 1949), and in U.S.A. the same virus

is transmitted by the bean leaf beetle, *Ceratoma trifurcata* (Smith, 1924).

Properties: The thermal inactivation point is between 65° and 70°C, the dilution end-point ranges between 1:10,000 to 1:100,000 and the longevity *in vitro* is twenty-four hours to twenty days. The virus is polyhedral measuring 24 to 27 nm for particles containing nucleic acid and 23 to 25 nm for empty particles.

The virus is serologically related to bean pod mottle virus and a distant relationship has been observed with a Dutch isolate of red clover mottle virus (Agrawal, 1964).

Host range: The virus infects the following hosts: *Arachis hypogaea*, *Cassia tora*, *Chenopodium amaranticolor*, *C. quinoa*, *Crotolaria striata*, *C. usaramensis*, *Dolichos lablab*, *Glycine max*, *Comphrena globosa*, *Phaseolus aureus*, *P. lunatus*, *P. mungo*, *P. vulgaris*, *Pisum sativum*, *Vigna unguiculata* and *Zinnia elegans* (Agrawal, 1964).

A beetle transmitted mosaic disease of cowpea in Cuba has been reported recently by Kvicala *et al.* (1973).

Transmission: The disease is transmitted by a bean leaf beetle, *C. ruficornis*. High concentrations of the virus are recorded from excrement of infective beetles.

Properties: The virus is found to have a thermal inactivation point of 65°C and a dilution end-point between 1:500,000 and 1:700,000 and a longevity *in vitro* in crude sap, diluted 1:10, of ten to fourteen days at a temperature of 20 to 22°C.

Host range: The virus is transmitted to eighteen species of leguminous and two species of non-leguminous plants.

Mosaic (India)

Capoor and Varma (1956) reported a mosaic disease of *Vigna cylindrica* from Poona and later Nariani and Kandaswami (1961) reported the virus on *V. sinensis* at Delhi.

Symptoms: The infected plants are characterised by mosaic mottling of leaves accompanied by distortion and reduction of the leaf size. The infected plants bear only a few pods. The pods are usually small and shrunken, containing only a few shrivelled seeds.

Transmission: The virus is sap transmissible. It is also transmitted by *Aphis medicaginis*, *A. craccivora*, *A. cevonymi*, *A. gossypii* and *Myzus persicae*. It is carried in about four per cent seeds of

V. cylindrica and also in *V. sinensis*.

Properties: The thermal death point of the virus lies between 85° to 90°C for ten minutes exposure. Even higher thermal inactivation point is found by Nariani and Kandaswamy (1961). Dilution end-point lies at 1:50,000 but not at 1:100,000. The virus loses its infectivity on storage at 24°C for nineteen days (Capoor and Varma, 1956). The virus is inactivated by desiccation at room temperature for six days (Nariani and Kandaswami, 1961).

Host range: The virus also infects, *Vigna sesquipedalis*, *Phaseolus lunatus* and *Canavalia ensiformis*, *Crotalaria juncea*, *Glycine max*. The virus produces local lesions on inoculated leaves of *Vigna vexillata*.

Control: Use virus free seeds. Varieties Suwwanee, Early sugar crowder and Taylor of *Vigna cylindrica* are tolerant.

Another aphid transmitted, cowpea mosaic disease was reported in 1968 by Chenulu *et al.*, from Delhi.

Symptoms: The symptoms consist of typical mosaic mottling, yellowing, reduction and distortion of leaf lamina. The symptoms are seen as small chlorotic patches on the primary leaves of plants arising from diseased seeds. There is a tendency of the affected leaves margins for curling downwards and cupping of the leaf.

Transmission: The disease is transmitted by sap, seed and aphids. Both *Aphis Craccivora* and *A. gossypii* are equally efficient in transmitting the virus.

Properties: The virus has a thermal death point of 55°C, dilution end-point of 1:500-1:1000, longevity *in vitro* of six hours at room temperature (25-30°C), forty-eight to seventy-two hours at frigidare temperature (6-8°C). The virus remains infective in phosphate buffer of pH 4.5 to 9.0 and was highly stable at pH 3.5. The isoelectric point was near pH 4.5.

The virus is antigenic and the particles are spherical with an average diameter of 23 nm.

Host range: The virus has a restricted host range. It infects *Crotalaria juncea*, *Phaseolus vulgaris* (vars. Ashgar bean, Bangalore local, Canadian Red, College early, College Pride, Stringless green pod, Stringless wax and Shinning Farm), systemically producing mosaic mottle. It also infects other *Vigna* species such as *V. pariflora*, *V. putigera*, *V. sesquipedalis*, *V. unguiculata* and *V. vexillata* producing mild chlorotic mottle or severe mosaic symptoms. Besides leguminosae it infects *Chenopodium amaranticolor* and

C. album producing distinct necrotic lesions.

The virus is found to be of close resemblance to cowpea mosaic viruses of Mclean (1941), Yu (1946) and Snyder (1942).

Nene and Shankar (1972) reported from Pantnagar, cowpea mosaic virus causing mosaic disease of *Vigna sinensis*.

Symptoms: The disease is characterised by mosaic mottling, vein-banding, puckering and distortion. Also blistering and bleaching of the leaves occur due to severe infection. The pods get curved, twisted and reduced in size. The seeds in such pods are shrivelled and lesser in number.

Transmission: The virus is mechanically transmissible. Three species of aphids, namely, *A. craccivora*, *A. gossypii* and *Myzus persicae* are the vectors of the virus.

Properties: The thermal inactivation point of the virus is 75°C. The dilution end-point of the virus is between 1:1000 and 1:5000. The longevity of the virus *in vitro* is fifty-six hours at 70–90°F and four days in refrigerator at 46.4°F.

Host range: Its host range is restricted to the family leguminosae except *Chenopodium amaranticolor* on which the virus produces necrotic local lesions.

Cowpea Mosaic Virus (Ceylon)

Abeygunawardena and Perera (1964) reported a cowpea mosaic virus from Ceylon. The virus is transmitted by *Aphis craccivora* but is not seed-borne. It has a thermal inactivation point between 55° and 60°C, dilution end-point of 1:3000 and longevity *in vitro* of one to two days. *Euphorbia geniculata* is a potential reservoir of the virus.

Cowpea Mosaic Viruses (South Africa)

Two mosaic viruses cowpea mosaic virus A and B affecting cowpea in South Africa have been described by Klessner (1960).

Cowpea Mosaic Virus A

Symptoms: Virus causes stunting and small malformed leaves with dark green vein-bands or mosaic with necrosis.

Transmission: The virus is mechanically transmitted. The vector is *Aphis craccivora*.

Properties: The thermal inactivation point is 62 to 65°C, the dilution end-point is 1:2000 and the longevity *in vitro* is two to four days.

Host range: The virus is restricted to the leguminosae.

Cowpea Mosaic Virus B

Symptoms: The disease is characterised by a dark green vein banding milder than that of virus A.

Transmission: Virus is mechanically transmitted. The vector of the virus is not known.

Properties: The thermal inactivation point is 60° to 62°C, the dilution end-point is 1:1000 and the longevity *in vitro* is two to three days.

Black Eye Cowpea Mosaic Virus (Florida)

Anderson (1955) described a cowpea mosaic virus from Florida.

Transmission: The virus is transmitted by sap inoculation and is seed borne. It is transmitted by *Aphis gossypii*, *Macrosiphum solanifolli* and *Myzus persicae*.

Properties: The virus has a thermal inactivation point 60–65°C, a dilution end-point of 1:10000 and longevity *in vitro* of one to two days.

Host range: The virus causes systemic infection in plants of genera, *Vigna*, *Phaseolus*, *Vicia*, *Crotalaria*, *Desmodium* and local infection in *Glycine max*, *Dolichos*, *Cassia*, *Zinnia* and *Chenopodium*.

Yellow Mosaic (Indonesia)

Symptoms: The disease is characterised by the presence of yellow mosaic on the leaves. The patches are found on both sides of the veins which are mostly chlorotic. The chlorotic parts are yellow coloured as compared to the common mosaic (Semangoen, 1958).

Transmission: The virus is transmitted by *Aphis medicaginis*.

Host range: Transmission experiments were attempted with this vector to *Arachis hypogaea*, *Psophocarpas tetragonolobus*, *Phaseolus lunatus*, *P. vulgaris*, *P. radiatus*, *Crotalaria juncea*, *C. striata*, *C. retusa*, *Soja max*, *Cajanus cajan*, *Canavalia ensiformis*, *Dolichos lablab*, *Pisum sativum*, *Nicotiana tabacum*, *N. glutinosa*, *Lycopersicon esculentum*, *Cucumis sativus* and *Ageratum conyzoides* but the results were negative.

Only *P. lunatus* and *P. radiatus* are found to be susceptible.

PIGEON PEA (*Cajanus cajan*)

Mosaic

Symptoms: Symptoms are confined to the trifoliate leaves of the upper branches. On young leaves, faint, scattered, green and yellow spots gradually develop into a severe mosaic. Small poorly developed green pods producing shrivelled seeds are found on heavily infected plants (Bisht and Banerjee, 1965).

Transmission: The disease is transmitted to pigeon pea by grafting but not to cowpea, *Phaseolus aureus* or *P. mungo*. The disease has been reported from Uttar Pradesh, India.

Sterility

Symptoms: The disease is characterised by pale green colour of the plants and absence of flowering branches, rendering them sterile. The leaves also display a distinct mosaic pattern (Capoor, 1952).

Transmission: The sterility disease of pigeon pea is transmitted by an eriophyid mite *Aceria cajani* (Seth, 1962). The virus is graft transmissible and is restricted to pigeon pea.

Properties: Leaves infected with the virus showed a chlorophyll reduction of up to 60.9 per cent a decrease in carotene, xanthophyll and total carbohydrate content and an increase in chlorophyll activity (Narayanaswamy and Ramakrishnan, 1965). Calcium, potassium, sodium and magnesium contents were lower in diseased than in healthy plants (Nambiar and Ramakrishnan, 1969a). Total N was higher in diseased than in healthy leaves at all ages (Nambiar and Ramakrishnan, 1969b). Total carbohydrates were significantly less in diseased than in healthy plants. Increased respiration of diseased plants was accompanied by a general reduction in organic acid content, but accumulation of citric and succinic acids, respectively was noted in the stem and root (Narayanaswamy and Ramakrishnan, 1966).

Control: The disease could probably be controlled by the removal of old and volunteer plants well in advance of the next sowing season (Seth, 1965).

Mung (*Phaseolus aureus*)

Yellow Mosaic

Symptoms: The disease is characterised by bright yellow patches

on the leaves interspersed with green areas and slight puckering. Leaf area is generally not reduced. The infected plants bear very few flowers and pods. The seed production is also affected (Nariani, 1960).

Transmission: The disease is graft transmissible and is transmitted by whitefly, *Bemisia tabaci* only, but not through sap.

Host range: *Phaseolus* spp., *Dolichos biflorus*, *Soja max*, *Cajanus cajan*, *Eclipta alba*, *Xanthium strumarium* and *Brachiaria ramosa* act as source of infection (Nariani, 1960; Nene, et al., 1971).

Control: The following insecticide plus mineral oil spray has been recommended (Nene, 1972). Malathion 0.1 per cent or Thiodan 0.1% + 0.1 per cent Metasystox + 2 per cent orchard oil four sprays at ten days interval starting from twentieth day after sowing give good control of the disease.

Leaf Curl

Symptoms: The disease is characterised by the appearance of chlorosis around some lateral veins and its branches along the margin of leaves. The leaves show curling of margin although rolling up of a few affected young leaves is not uncommon. Some of the leaves show twisting. These leaves become brittle and veins show reddish brown discolouration on the under surface which also extends to the petiole. Affected plants remain stunted.

Transmission: The virus is graft transmissible and it is also transmitted mechanically (Nene, 1972).

SOYBEAN (*Soja max*)

Mosaic

Symptoms: The disease is characterised in the beginning by the appearance of yellowish vein clearing of newly developed leaves, later changing into alternate light and dark green patches. Leaf area is reduced and due to upward proliferation of the blades leaves look puckered. Diseased plants look stunted and set fewer pods than the healthy. High temperature mask the symptoms of the virus (Canover, 1948).

Transmission: The disease is transmitted through seed and by aphid vectors, such as *Aphis gossypii*, *A. craccivora*, *A. evonymii* and *Myzus persicae*. It is also transmitted mechanically (Nariani and Pingaley, 1960).

Properties: The virus is inactivated when heated to 62°C but not at 60°C. The virus sap remains infective upto a dilution of 1:1000 but not at 1:5000. The virus loses its infectivity by storage for four days at room temperature. The particle size is 650–725 nm \times 15–18 nm (Galvez, 1963).

Host range: The virus is restricted to soybean only and has been reported from India

Another virus transmitted* to soybean is 'Yellow mosaic' of *Phaseolus aureus* (Nariani, 1960).

Control: The mosaic disease can be controlled by using the virus free seed.

URID (*Phaseolus mungo*)

Mosaic

Symptoms. The disease is characterised by a mosaic pattern of broad, light green areas and blistering of leaf blade. The virus induces retardation of the growth of the affected plants which usually shed most of their flowers.

Transmission: The virus is readily transmitted by sap inoculation. Sahare and Raychaudhuri, 1963).

Properties: The virus is completely inactivated by heating at 50 C for ten minutes, but not at 55°C and at a dilution of 1:2,000. It retains infectivity during storage at room temperature 25–30°C for twenty-four hours (Sahare and Raychaudhuri, 1963).

Host range: The host range of the virus is found to be confined to the family leguminosae, *Phaseolus mungo*, *P. aureus*, *Cyamopsis tetragonoloba*, *Dolichos biflorus* (Srivastava *et al*, 1969). The virus resembles the common bean mosaic virus (Sahare and Raychaudhuri, 1963) and occurs commonly in India.

Two other viruses that have so far been transmitted to urid are 'yellow mosaic' of *Phaseolus aureus* and mosaic of *Vigna sinensis*, the latter being sap transmissible. Two lines of Urid bean UPU-1 and UPU-2 have been reported to be tolerant to yellow mosaic (Nene, 1972).

Leaf Crinkle

The disease has been reported to occur in India.

Symptoms: Generally the third trifoliate leaf shows the symptoms first and, an increase in the size of leaves and lighter green colour

is observed. After that, typical leaf crinkly becomes more conspicuous within a week, the mild and transitory symptoms on 2nd trifoliate leaf start appearing. As the infected plant grows further, the crinkling and rugosity on older trifoliates goes on decreasing.

Symptom expression is influenced by the age at which plants get infected.

Transmission: The virus is sap transmissible, can be transmitted through the seed (Kolte and Nene, 1970). *Aphis craccivora* has been found to be the vector (Dhingra, 1974).

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21

Spices

CARDAMOM (*Elettaria cardamomum*)

Small Cardamom

The plantations are known to be affected by a most destructive disease, locally known as 'Katte' (death) or marble disease in Karnataka and Kerala States of India.

Katte disease: The 'Katte' disease or mosaic virus attacking *Elettaria cardamomum* at all stages of growth is characterised by the general chlorosis of entire leaf having broken stripes of pale green colour evenly distributed over the leaf surface and sometimes also on the leaf sheaths. In older plants growth and production of flowering spikes are retarded (Capoor, 1967).

Transmission: The disease is not transmitted by sap, seed or soil (Varma and Capoor, 1958). In nature it is transmitted by the banana aphid, *Pentalonia nigronervosa*. The virus is semi-persistent. The aphids are commonly found on banana during winter months. The disease is transmitted through the aphids to cardamom at all stages of growth, except in very young seedlings, where the aphids do not colonise (Uppal *et al.*, 1945; Varma, 1956).

Host range: The virus is readily transmitted to *Amomum cannaecarpum* only (Capoor, 1967, Varma and Capoor, 1958).

Control: By eradication of affected clumps of small cardamom; use of virus free seedlings raised from true seed gave encouraging results.

Large Cardamom (*Amomum subulatum*)

Two diseases have been recorded on large cardamom plants, namely, 'Chirke' (mosaic streak) and 'Foorkey' (bushy

dwarf) from Darjeeling district of India.

'Chirke' (*Mosaic streak*)

The disease is characterised by mosaic streaks on leaves which gradually coalesce and eventually turn brown. The leaves ultimately dry up and wither. Flowering in diseased plants is reduced and only one to five flowers develop in one inflorescence as against sixteen to twenty flowers borne by healthy plants (Raychaudhuri and Chatterjee, 1958).

Transmission: The causal virus is sap transmissible and is also spread through the agency of *Rhopalosiphum maidis* (Raychaudhuri and Chatterjee, 1961), *Brachycaudus helichrysi* and an unidentified aphid collected from squash (Raychaudhuri and Chatterjee, 1965). The virus is transmitted non-persistently from cardamom to cardamom and to wheat (Raychaudhuri and Chatterjee, 1965).

A single aphid of *R. maidis* requires an acquisition feeding period of ten minutes for transmitting the disease.

Properties: The virus can tolerate exposure upto 50°C for ten minutes but gets innocuous when exposed to 60°C. The virus withstands dilution upto 1:2000 but is rendered inactive at 1:5,000. Longevity *in vitro* is four to eight days at room temperature ranging from 4.4. to 33.3°C (Raychaudhuri and Chatterjee, 1965; Raychaudhuri and Ganguly, 1965).

The large cardamom varieties, Ramnok Ramsai, Kati, Hario, Churampha, the wild cardamom (*Amomum aromaticum*) arrow root (*Curcuma angustifolia*), the perennial weed (*Acorus calamus*) and eleven varieties of ginger have been found to be susceptible (Raychaudhuri and Chatterjee, 1964; Raychaudhuri and Ganguly, 1965).

Control: Roguing of the affected clumps should be carried out for a number of years. Disease free seedlings raised from true seeds should be used for cultivation.

The virus has been observed to infect wheat plants cultivated in the vicinity of virus affected cardamom plants. Cardamom varieties Sawney and Kopringer and the wheat-varieties Ridley, NP 803 and NP 809 are resistant to the virus.

Foorkey (*Foorkey means bushy dwarf or stunt*)

Symptoms: The affected plants produce many stunted shoots which fail to produce flowers. The leaves are small, slightly curled

and pale green in colour (Varma and Capoor, 1964; Vasudeva, 1956). Small and wild cardamom plants are susceptible to the virus. In this case, the inflorescence during third year of growth becomes stunted, thereby producing no flowers and fruits (Raychaudhuri and Chatterjee, 1958).

Transmission: The disease is not transmitted mechanically through sap, but aphids, *Pentalonia nigronervosa* (Vasudeva, 1956) and *Micromyzus kalimpongensis* transmitted in a persistent manner. The aphids infest *Hedychium corogarium* and also colonise on *Amomum subulatum* in Kalimpong area (Basu and Ganguly, 1968). **Host range:** The cardamom variety Kopringle is found to be tolerant to the virus (Chattopadhyay and Bhowmik, 1965; Majumdar, 1966).

Control: The disease can be controlled by eradication of infected clumps, by injecting 40 per cent Agroxone-4 (Liquid), 2, 4-D herbicide in rhizomes, use of disease free seedlings raised from true seed, and spraying with 0.04 per cent Folidol-E 605 every three weeks during May to December (Chattopadhyay and Bhowmik, 1965).

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22

Vegetables

PIGWEEED (*Amaranthus* sp.)

Mosaic

Symptoms: Infected plants are characterised by mosaic mottling of leaves and yellowing of veins (Phatak, 1965; Govindaswamy *et al.*, 1967).

The virus occurs naturally on *A. blitum*, *A. viridis*, *A. gangeticus* and *A. caudatus* in India.

Transmission: The virus is sap transmissible. The aphids, *Myzus persicae*, *Aphis gossypii*, *A. craccivora* and the whitefly *Bemisia tabaci* failed to transmit the virus.

Properties: The virus has thermal inactivation point of 55–60°C dilution end-point between 1:10 and 1:100 and longevity *in vitro* of eight to twenty-four hours (Govindaswamy *et al.*, 1967).

Host range: The virus infects species of *Amaranthus*, *Nicotiana tabacum* var. White Burley and *N. glutinosa*.

AMORPHOPHALLUS (*Amorphophallus campanulatus*)

Mosaic

Symptoms: The affected plants are dwarfed and become chlorotic in appearance. The mosaic mottling tends to be intense in leaves reaching maturity. Some affected plants show distortion of leaf lamina also in addition to mottling. The disease has been reported from India.

Transmission: The virus is not mechanically transmitted, but is readily transmitted by *Myzus persicae* and *Aphis gossypii* (Capoor and Rao, 1969).

Host range: The virus is transmitted to *Amaranthus tricolor*, *Atriplex hortensis*, *Cyamopsis tetragonoloba* and *Phaseolus vulgaris*, but not to *Colocasia esculenta* (Capoor and Rao, 1969).

BEET (*Beta vulgaris*)

Purple Leaf

Symptoms: The virus causes mild chlorotic spots in the beginning but later on the entire affected leaf gets puckered and curled or rolled. Young developing leaves become intense purple in colour and brittle in texture.

Transmission: The virus is transmitted by sap inoculation.

Properties: The virus withstands heating for ten minutes at 91° C but not at 95° C. It tolerates dilution upto 10^{-5} but not to 10^{-6} and stands ageing for over a month. The virus resembles TMV and has been reported from India.

Host range: The virus infects following species of plants: *Cassia tora*, *Nicotiana glutinosa*, *Chenopodium amaranticolor*, *C. album*, *N. tabacum* cv. White Burley, *N. sylvestris*, *Lycopersicon esculentum*, *Gomphrena globosa* and *Spinacia oleracea* (Capoor and Sharma, 1965).

Curly Top

Symptoms: The disease is characterised by veinlet clearing, vein swelling and leaf rolling of the infected plants. The first affected leaves are curled with translucent veinlets and the petioles are shorter than on healthy plants. Phloem necrosis is characteristic of the curly top virus. Severity of leaf symptoms is due to vascular phloem necrosis (Lawcett, 1925, Bennett *et al.*, 1946). The disease has been reported from Argentina.

Transmission: The virus is transmitted through insect vector *Agalliana ensigera*.

Host range: The virus infects marigolds var. Golden globe, *petunia* sp., zinnia and chickweed.

Curly Top

Symptoms: The disease is characterised by development of vein clearing usually throughout the veins of young leaves but sometimes confined to circular spots and lines. The vein clearing is accompanied by rugosity and downward rolling of the leaf edge. As

the leaf matures, the cleared portions turn normal green and the veins become swollen and irregular. Sometimes small spike like outgrowths are produced. The leaves are normal in shape and the plants show very little stunting. The disease has been reported from Brazil.

Transmission: The virus is not mechanically transmissible and has a specific leafhopper vector, namely, *Agalia albidula* (Bennett and Costa, 1949).

Host range: The virus infects following plant species:

Spinacia oleracea, *Nicotiana tabacum*, *Lycopersicon esculentum*, *Acanthospermum hispidum* and *Stellaria media*.

Yellow Vein

Symptoms: The disease is characterised by appearance of yellowing of leaf veins, dwarfing of one side of the plant in early stages of infection.

The disease has been reported from Argentina.

Transmission: The virus is transmissible by grafting only. It is not seed-borne. It has no vectors. The disease is not transmitted by *Cuscuta* spp. (Bennett, 1956).

BEAN (*Phaseolus vulgaris*)

Atypical Mosaic

A virus causing atypical mosaic on bean and sunnhemp appears to be related to TMV.

Symptoms: The symptoms consist of vein clearing followed by chlorotic spots. Vein banding and mosaic symptoms appear in later stages. Reduction and deformation of leaves are common. Puckering of the leaves takes place in some plants. Infected plants become stunted. The disease has been reported from India (Nagaich and Vashisth, 1963).

Transmission: It is only sap transmissible.

Properties: It has a dilution end-point of 1:1000 and is inactivated at 93–94°C. It has a longevity of forty-five days at room temperature, ninety days at 0°C. The virus is stable in the range of pH 3 to 8 (Nagaich and Vashisth, 1963).

Mosaic

Symptoms: The disease is characterised by mosaic mottling of

leaves, blistering and downward curling of the lamina. The plants become stunted, dwarfed and bushy in appearance. Shedding of flowers is observed in the infected plants. The setting of the pods is also delayed.

Transmission: The virus is transmitted through seeds of infected plants and spreads in the field through *Aphis craccivora*, *A. gossypii*, *A. evonymi* and *Myzus persicae* (Yaraguntaiah and Nariani, 1963).

Host range: *Phaseolus mungo*, *P. aureus*, *P. lunatus*, *P. calcaratus*, *Trigonella foenum-graecum*, *Lupinus albus*, *Cicer arietinum*, *Cyamopsis tetragonoloba*, *Crotalaria sericea* and *Vicia faba* are infected by this virus. Bea var. Kentucky Wonder has proved to be immune to infection.

Yellow Mosaic

Symptoms: The disease is characterised by the development of slightly irregular and small light yellow spots in the dark green background of leaflets. The yellowing gradually spreads over the entire surface, causing the leaflets to become more or less chlorotic. Symptoms do not become masked in later stages of growth. Plants become decidedly stunted and bushy because of a reduction in the length of the internodes and a proliferation of branches. Maturity is delayed and production of pods greatly reduced (Pierce, 1934).

Transmission: The virus is sap transmissible and infection is easily rendered by the addition of carborundum powder to the inoculum. The insect vectors are *Acyrtosiphum pisum*, *Aphis fabae*, *Megoura viciae* and *Myzus persicae*.

Properties: Thermal inactivation point ranges between 56–60°C when exposed for ten minutes. Dilution end-point ranges between 1:800 to 1:1000 and ageing *in vitro* twenty-four to thirty-two hours at room temperature. The virus particle is flexuous rod measuring 750 nm in length and 13 to 14 nm in width.

Host range: It infects *Tetragonia expensa*, *Crotalaria spectabilis*, *Gladiolus* sp., *Pisum sativum*, *Melilotus alba* and *Vicia faba*.

Seventy-seven leguminous and three non-leguminous species (Opium poppy, *Cirsium arvense* and *Chenopodium album*); Soybean, *Soja max* Piper are found to be its natural hosts (Kovachevsky, 1968).

BROAD BEAN (*Vicia faba*)**Mosaic**

Symptoms: The disease is characterised by clearing of veins and later severe mosaic mottling, slight puckering and smalling of the young leaves and stipules (Azad *et al.*, 1961).

Transmission: The causal virus is sap transmissible but is not transmitted through seed. *Aphis craccivora*, *A. rumicis*, *Macrosiphoniella sonbornii* and *Myzus persicae* are reported to be the vectors of the virus (Azad *et al.*, 1961).

Properties: The virus has a thermal inactivation point of 55–60°C, a dilution end-point between 10^{-3} and 10^{-4} , longevity *in vitro* of 120 to 144 hours at 10–15°C and optimum pH for infection ranged from 8 to 10.

Host range: The virus can be transmitted to *Vicia faba* vars. Beck's Green Gen., Broad Taylor's and Sutton's Early, *V. sativa*, *V. villosa*, *V. tetrasperma*, *Glycine max*, *Lupinus leuteus*, *L. albus*, *L. texanus*, *Lathyrus odoratus*, *L. sativus*, *Medicago hispida*, *Melilotus alba*, *Trigonella foenum-graecum*, *Trifolium incarnatum*, *T. repens*, *T. pratense*, *Pisum sativum* (Azad *et al.*, 1961).

Nour and Nour (1962a) reported a mosaic disease of broad bean from Sudan caused by pea mosaic virus which is described as follows:

Symptoms: The disease is characterised by mosaic, stunting and flower or pod shedding with severe yield reduction.

Transmission: The virus is transmitted by aphids and is not found to be transmitted through seeds of broad bean.

Properties: The causal virus loses its infectivity when heated for ten minutes at 65°C, but not at 60°C, has a dilution end-point of 1:6000 and longevity *in vitro* of forty-eight hours at 20–25°C. Electron microscopy revealed long flexuous threads measuring approximately 725 nm.

Nour and Nour (1962b) also reported broad bean to be naturally infected with the alfalfa mosaic virus, which is described as follows:

Symptoms: The virus causes local necrotic lesions in the beginning, but later induced stem necrosis which often caused wilting and death of the plant.

Transmission: The virus is transmitted through *Aphis gossypii*.

Properties: The virus has a thermal inactivation point of 60–75°C,

a dilution end-point between 1:200 to 1:2000, and longevity *in vitro* of one to two days at 25°C

Host range: The host range of the virus included members of the families leguminosae, cucurbitaceae, compositae

COMMON BEAN (*Dolichos lablab*)

Enation Mosaic

Symptoms. The disease is characterised by the malformed leaves, due to reduction in leaf lamina and production of foliar enations on the under surface (Capoor and Varma, 1948a)

Transmission: The virus is sap transmissible.

Properties: The dilution end-point of the virus in crude sap lies between 5×10^{-6} and 3×10^{-6} . It can resist ten minutes heating at 90° C but is inactivated at 95° C. Its virulence is not impaired after six years storage in the laboratory. The virus is serologically related to the Nigerian cowpea mosaic virus, a strain of TMV (Bawden, 1958)

Host range: The virus from *Dolichos lablab* can infect large number of other legumes including beans (*Phaseolus vulgaris*) and White Burley tobacco (Capoor and Varma, 1948a)

Yellow Mosaic

Symptoms. The disease is characterised by the bright yellow patches on the leaves without any distortion (Capoor and Varma, 1950a). Symptoms start as faint discoloured patches or leaves about fourteen to twenty days after inoculation which gradually increase in size leaving only a few spots and stripes of green tissue.

Transmission: The virus is transmitted through the whitefly, *Bemisia tabaci*

DOUBLE BEAN (*Phaseolus lunatus*)

Yellow Mosaic

Symptoms: The leaves of the infected double bean plants develop bright yellow patches and the diseased plants stand out distinct

Transmission: The disease is transmitted by whitefly, *Bemisia tabaci*.

Host range: The virus also affects *P. limensis*, *P. vulgaris*, *P. aureus*, *Dolichos biflorus* and *Canavalia ensiformis* (Capoor and Varma, 1948b).

RUNNER BEAN (*Phaseolus multiflorus*)**Mosaic**

Symptoms: The disease is characterised by mosaic mottling and slight deformation of the secondary leaves. The diseased plants are stunted (Vashisth and Nagaich, 1965).

Transmission: The disease is transmitted by sap inoculation. The virus is seed-borne and 42 per cent of the seeds of infected plants transmit the disease (Vashisth and Nagaich, 1965). *Aphis craccivora* and *Myzus persicae* failed to transmit the virus.

Properties: It is inactivated by dilution beyond 1/6,000 and heating above 63° C for ten minutes. *In vitro* it is viable for sixty-six days when frozen in frigidaire but inactivated between twenty to twenty-two days when stored at room temperature (15 to 21°C).

Host range: The disease is transmitted to *Phaseolus vulgaris*, var. French yellow, Black negro and *Fesbania aculeata*. Ten varieties of *Phaseolus vulgaris* and *Lathyrus sativus* were found to be symptomless carriers.

BHINDI (*Abelmoschus esculentus*)**Yellow Vein Mosaic**

Symptoms: The disease is characterised by the vein clearing followed by veinal chlorosis of the leaves. The yellow net work of veins is very conspicuous and the veins and veinlets are thickened. In severe cases the chlorosis may extend to the interveinal areas and may result in complete yellowing of the leaves. The fruits produced on diseased plants are often malformed, pale in colour and tough in texture (Capoor and Varma, 1950b). The disease is prevalent in India and Ceylon.

Transmission: The virus is not transmitted through sap or seed or by dodder (*Cuscuta reflexa*). It is, however, readily transmitted by grafting and by the whitefly, *Bemisia tabaci*, but not through the eggs to offspring of viruliferous insects (Capoor and Varma, 1950b).

Host range. The virus infects several species of *Hibiscus* and *Abelmoschus* and *Althea rosea*. *Abelmoschus esculentus*, *A. moschatatus* and *A. ficulneus* produce typical yellow vein mosaic symptoms. *Abelmoschus manihot*, *A. tuberculatus*, *A. angulosus*, *Hibiscus cannabinus* and *H. subdariffa* produce vein swellings on the under surface of the leaves (Nariani and Seth, 1958).

Control: The plants *Abelmoschus manihot* var. *Pungens*, *A. crinitus*, *Hibiscus vitifolius* and *H. panduræ-formis* have been shown to be immune to the virus (Nariani and Seth, 1958). The disease can be controlled by removal of malvaceous seed hosts of the virus and eradicating the vector population by spraying with insecticides (Capoor and Varma, 1950b).

The variety Pusa sawani is reported to be tolerant to the disease (Joshi *et al.*, 1960).

BRINJAL OR EGGPLANT (*Solanum melongena*)

Mosaic

Symptoms: Naturally infected plants show a mosaic of light and dark green areas. This disease is reported from Trinidad and South America.

Transmission: The virus is transmitted by mechanical means and the vector is a beetle, *Epitrix* sp. (Dale, 1954).

Properties: The thermal inactivation point is 78°C. The particles are isometric and measure 30 nm in diameter and have thirty-two major morphological subunits (Gibbs and Harrison, 1969; Kitajima and Costa, 1968).

Host range: *Nicotiana glutinosa*, *N. delevelandii*, *Chenopodium quinoa* and *C. amaranticolor*.

In India Kulkarni (1924) was first to record a mosaic of brinjal from Bombay State but he did not give any details. Raychaudhuri (1947) described a mosaic disease of brinjal from Delhi. The symptoms included mosaic mottling, puckering and crinkling of the leaves. The disease could be transmitted by grafting only.

Capoor and Sharma (1961) and Sharma (1969) reported the occurrence of five sap transmissible viruses in eggplant from Poona of which one is identified as tobacco mosaic virus and another as cucumber mosaic virus.

A new brinjal mosaic virus believed to be a strain of cucumber mosaic virus has been described from Delhi which is as follows (Seth *et al.*, 1967).

Symptoms: Younger leaves of infected plants show pronounced mosaic mottling and the older leaves show only mild mottling. Various types of deformities like puckering, crinkling and blisters of various sizes may develop on the severely infected leaves. The

diseased plants bear less flowers and distorted fruits of comparatively smaller size.

Transmission: The virus is transmitted by aphids, namely, *Aphis gossypii*, *Myzus persicae* and *Aphis craccivora* and is also sap transmissible.

Properties: The purified virus particles under electron microscope have been found to be spherical in shape, 35–36 nm in diameter (Seth and Raychaudhuri, 1973).

Host range: The virus can be readily transmitted by sap inoculation to *Solanum gilo*, *S. indicum*, *S. integrifolium*, *S. khasianum*, *S. sisymbriifolium*, *Cacciria cordifolia*, *Cucumis anguria*, *C. melo* var., *Utilissimus*, *Cucurbita maxima*, *C. pepo*, *Lagenaria siceraria*, *Spinacia oleracea*, *Calendula officinalis*, *Carthamus tinctorius*, *Zinnia elegans*, *Ocimum sanctum*, *Salvia officinalis*, *Tropaeolum majus* and *Beta vulgaris* cv. *Benghalensis*.

Verma and Rashmi Lal (1967) described a mosaic disease of brinjal. The virus resembled tobacco etch virus in its host range and symptoms; and was transmitted by *Myzus persicae*.

CABBAGE (*Brassica oleracea* var. *Capitata*)

Black Ring Spot

Symptoms. The virus first induces small yellow spots on the leaves and then as the leaves become older, the spots enlarge and the tissue round the spots dies and becomes almost black in colour, giving the effect of black ring spots. The latter markings are more noticeable when the plants are fully grown and occur mainly on the old outermost leaves. When the spots are numerous, the areas between them may finally turn yellow and dry, presenting an appearance of scorching (McClean, 1952). The disease has been reported from South Africa.

Transmission: The virus is sap transmissible.

Host range: Cabbage ring spot not only attacks crucifers like cabbage and cauliflower, brussels sprout, sprouting broccoli, curly kale, turnip and stock, but also some poppy species and endive lettuce (McClean, 1952). It also attacks Egyptian Radish (*Raphanus sativus* var. *Aegyptiacus*) as reported by Eskarova Josephine (1966).

A strain of cabbage black ringspot virus has been isolated from some ornamental plants like *Iberis umbellata*, *Matthiola incana*

and *Hesperis matronalis* from India (Bhargava and Joshi, 1960).

CAULIFLOWER (*Brassica oleracea* var. Botrytis)

Mosaic

Symptoms: In the beginning the disease affects the veins in the young leaves and forms a yellow net-work and thereafter dark green bands develop along the sides of the veins. Plants attacked at an early age remain stunted.

Transmission: The virus can be transmitted artificially by sap inoculation. Two species of aphids, *Myzus persicae* and *Brevicoryne brassicae* can transmit the virus.

Host range: The disease is only known to attack the crucifers and in addition to cabbage and cauliflower, it has been recorded in Brussels sprouts, sprouting broccoli and stocks in South Africa (McClellan, 1952).

CHILLI (*Capsicum annuum*)

Leaf Curl

Symptoms: The chief symptoms produced are abaxial and adaxial curling of the leaves puckering, and distortion of the inter-veinal areas, and thickening and swelling of the veins. In advanced stages of the disease, axillary buds are stimulated to produce clusters of leaves which are reduced in size. The whole plant assumes a bushy appearance and stunted growth. Fewer flowers and fruits are developed on the diseased plants (Mishra *et al.*, 1963). It is one of the most serious diseases in India.

Transmission: The disease is transmitted by means of wedge grafting as well as by *Bemisia tabaci* from chilli to chilli.

Host range: The virus is successfully transmitted by means of whiteflies to *Nicotiana tabacum* cv. White Burley, *Lycopersicon esculentum*, *Nicotiana glutinosa*, *Petunia hybrida*, *Capsicum annuum*, *C. frutescens*, *C. microcarpum*, *C. sinensis*, *C. pubescens*, *C. pendulum* and *Crotalaria juncea*. The virus is reported to be identical with the tobacco leaf curl virus earlier reported by Pal and Tandon (1937).

Danraj and Seth (1968) described a severe strain of leaf curl characterised by curling of leaves, thickening of veins and enations

on the under surface of the leaves from Delhi.

Control: Chilli varieties Puri Red and Puri Orange are resistant (Mishra *et al.*, 1963). The disease can be controlled by spraying the crop with Parathion or Diazinon, which are effective against the whitefly. The crop must be sprayed right from the early stages of growth and the diseased plants, which appear, should be rogued out.

Mosaic

Mosaic disease of chilli is caused by at least seven viruses in India.

1. The chilli mosaic (Jha and Raychaudhuri, 1956)
2. Virus causing necrosis on chilli (Mishra, 1963)
3. *TMV* and strain of *TMV* (Kandaswamy *et al.*, 1963; Mathur *et al.*, 1966)
4. Cucumber mosaic virus (Anjaneyulu and Apparao, 1967)
5. The vein banding mosaic virus of chilli—a strain of potato virus Y (Joshi and Bhargava, 1962)
6. A strain of potato virus Y (Jeyarajan and Ramakrishnan, 1969)
8. Potato virus X (Rao *et al.*, 1970)

The most prevalent mosaic disease of chilli is the one reported by Jha and Raychaudhuri (1956) occurring under natural conditions on chilli crop (*Capsicum frutescens*).

Symptoms: The symptoms of chilli mosaic disease consist of mosaic mottling, distortion and filiformy of leaves. Slight curling, marginal rolling and stunting of leaves are sometimes seen. Severely affected plants produced few flowers and fruits (Jha and Raychaudhuri, 1956). Swaminathan (1956) found that the mosaic infection in chilli caused meiotic disturbances, delayed embryonic development and resulted in few seeds per fruit.

Transmission: The virus is transmitted by sap and *Aphis gossypii* (Jha and Raychaudhuri, 1956); *A. evonymi* and *Myzus persicae* (Nariani and Sastry, 1958). This virus is non-persistent in its vector and is transmitted equally efficiently by both alate and apterous forms (Nariani and Sastry, 1962). The virus is not carried through seed.

Properties: The virus has a thermal inactivation point of 55–56°C, a dilution end-point between 1:25,000–1:30,000 and longevity *in vitro* of fifteen to twenty-two days at room temperature.

Host range: In addition to chilli, the virus is transmitted by sap

inoculation to *Nicotiana tabacum*, *N. tabacum* cv. White Burley, tobacco selections 78 and 78A, *N. glutinosa*, *Solanum nigrum*, *Petunia hybrida*, *Cucumis sativus*, *C. melo* cv. Utilissimus and *Carthamus tinctorius*. Potato cv. President and Craig's Defiance as also *Datura stramonium* carry the disease symptomlessly.

Control: Chilli varieties Puri Red and Puri Orange are resistant (Mishra *et al.*, 1963).

Mishra (1963) studied a mosaic virus causing necrosis on chillies. The virus caused in addition to mosaic mottling veinal and stem necrosis in infected plants.

Transmission: The virus is transmitted by sap and by *A. gossypii*, *A. craccivora* and *Myzus persicae*.

Properties: The virus has a thermal inactivation point between 60–65°C, a dilution end-point between 1:500 to 1:1000 and longevity *in vitro* of one to two hours at 32–35°C and two to three days at 7–10°C.

Host range: The virus infects members of solanaceae, leguminosae and chenopodiaceae.

Anjaneyulu and Apparao (1967) described the occurrence of cucumber mosaic virus from Andhra Pradesh and Kandaswamy *et al.* (1966) reported the presence of strains of CMV from Tamilnadu.

Joshi and Bhargava (1962) studied vein banding mosaic virus—a strain of potato virus Y on chilli, is described as follows:

Transmission: The virus is transmitted by sap and by *M. persicae* and *A. gossypii* but not through seed.

Rao, *et al.* (1970) described a strain of Potato virus X as given below:

Symptoms: The affected plants are characterised by stunted growth and bushy appearance. Leaves are much reduced in size and malformed with various types of mosaic mottling. Fruit setting is much reduced, and when formed are reduced in size and malformed. Local necrotic lesions with white spot at the centre and surrounded by reddish band are also observed (Rao *et al.*, 1970).

Transmission: The virus is easily transmitted by sap inoculation. Aphids, *Aphis gossypii* and *Myzus persicae* failed to transmit the disease.

Properties: The virus is found to withstand heating upto 72°C. Exposure to temperature of 75°C. and above resulted in complete

loss of infectivity. The virus is found to have a dilution end-point of 10^{-5} to 10^{-6} and loses its infectivity between eighteen to twenty days of storage at room temperature (20–30°C).

Host range: The virus infected thirty-two plants belonging to ten genera and fourteen species distributed in Solanaceae, Chenopodiaceae and Amarantaceae. Based on symptoms, transmission, host range and physical properties the virus is identified as a ringspot strain of potato virus X (Rao *et al.*, 1970).

Pepper Vein Banding Mosaic (Florida)

Symptoms: The disease is characterised by vein clearing in the young leaves and vein banding which develops after about three weeks (Simon, 1956).

Transmission: The virus is transmitted through sap and *Myzus persicae* Sulz. and *Aphis gossypii*. Transmission is one of the non-persistent type.

Properties: The thermal inactivation point ranges between 60–65°C, dilution end-point between 1:10,000 and 1:20,000.

Host range: The experimental host range is restricted mainly to solanaceous plants, pepper, *N. glutinosa*, *N. tabacum*, although the virus infects *Zinnia elegans* and *Portulaca oleracea*. On the other hand the virus failed to infect *Datura stramonium*.

The disease has been reported from Southern Florida, USA, Trinidad and Israel.

CUCURBITS

Cucumber Mosaic Virus or Cucumis Virus I

Symptoms: The disease is characterised by the presence of greenish areas and circular or elongated spots on the leaves. Growth of the infected plants become stunted and internodes shortened leaves reduced to half their normal size. Fruits misshapen. The disease is of world wide distribution.

Transmission: The disease is transmitted through seeds of *Cucurbita maxima* (Mukhopadhyay and Saha, 1968). Virus is sap transmitted and its vectors are *Aphis gossypii*, *A. cracchvora*, *A. evonymi* and *Myzus persicae*.

Properties: The virus has an inactivation point of 60 to 70°C, dilution end-point of 1:10,000 and longevity *in vitro* of 72–96 hours at room temperature.

Host range: Virus has a very wide host range which includes *Nicotiana tabacum*, *N. glutinosa*, *Cucumis sativa*, *C. melo* cv. *Utilissima*, *C. anguria*, *Cucurbita moschata*, *Duchesne*, *C. pepo*, *C. maxima*, *Luffa acutangula*. It infects monocots also, *Commelina nudiflora*, *Allium cepa*, *Zillium* sp. banana and maize, etc.

CMV is found in cucumber, marrow, muskmelon, fodder hosts, perivinkle, tomatoes, peppers and eggplants (Nizany and Wilkinson, 1961).

Mosaic of Snakegourd (*Tichosanthes arguina*)

The disease is caused by cucumber mosaic virus or *Cucumis virus* 1 and has been reported from India (Shankar *et al.*, 1969).

Symptoms: The disease is characterised by a mosaic pattern of irregular dark green and yellow chlorotic patches on the lamina. The affected plants are stunted, produce fewer flowers and show leaf crinkling.

Transmission: Disease can be transmitted by mechanical inoculation and by the insect vectors, *Aphis gossypii* and *Myzus persicae*. *Nicotiana glutinosa*, *Chenopodium amaranticolor*, *C. guinoa* and *Cucurbita pepo*, among others can be infected with the virus.

Properties: The symptoms produced on the different hosts and the physical properties of the virus are in agreement with the cucumber mosaic virus. The purified virus preparations revealed spherical virus particles with a diameter of 29 nm (Shankar *et al.*, 1969).

Cucumber Green Mottle Virus or Bottlegourd Mosaic

Several viruses have been reported to cause mosaic disease in bottle-gourd in India. Vasudeva and Lal (1943) reported a mosaic disease of bottle gourd caused by *Cucumis virus* 3. Later, Capoor and Varma (1948c) observed that the mosaic of bottle gourd is caused by a strain of *Cucumis virus* 2 and designated it as 2b. Vasudeva *et al.* (1949) reported a new strain of *Cucumis virus* 2 and designated it as *Cucumis Virus* 2C. Later, Raychaudhuri *et al.* (1950) studied in detail the properties of the virus.

Symptoms: The symptoms are, irregular light green or dark-green mottling, occasionally with pale, yellow chlorotic area on the leaves.

Transmission: The virus is sap transmissible, but no vector has so far been determined.

Properties: The virus has a thermal inactivation point of 95–98°C, a dilution end-point of 1:10,000, longevity *in vitro* for more than a year. The electron micrographs of purified virus preparations revealed rigid rod shaped particles with a modal length of 280 nm and an average width of 17 nm. The purified virus preparation showed maximum UV absorption at 260 μ and four Schlieren peaks in analytical ultra centrifuge with $S_{20} W$ values of 134, 117, 89 and 64. It produced a single bluish light scattering virus zone in density gradient sucrose columns which contained the maximum infectivity and serological activity (Shankar *et al.*, 1971).

The antiserum was found to be specific, has a titre of 1:4096 and produced flagellar type of precipitate characteristic of rod shaped viruses in precipitin tests. It gave a single curved band in agar gel diffusion slide tests. The virus showed serological relationship with TMV (Shankar *et al.*, 1971).

Host range: Host range of the virus is restricted to family cucurbitaceae. Visible symptoms are produced on *L. siceraria*, *Cucurbita moschata*, *Cucumis sativus* (Vasudeva *et al.*, 1949). The disease is symptomlessly carried on *Datura stramonium*, *Luffa acutangula*, *Momordica charantia* and *Colosynthes vulgaris*.

PUMPKIN (*Cucurbita moschata*)

Mosaic

Symptoms: The disease consists of mosaic mottling of the leaves in the beginning. The younger leaves show complete chlorosis followed by green vein banding, whereas the older leaves show prominent dark green raised blisters, the rest of the leaf lamina being chlorotic. Sometimes leaves show chlorosis of veins and veinlets leaving interveinal areas green. The leaf lamina is very often distorted and is reduced to narrow strips of attendant leaf tissue. The veins and veinlets usually extend beyond the margins of narrow projections of varying sizes giving the leaves a filiform shape. In mild cases the flowering is delayed and the size of flowers is reduced. Very often vines do not bear any fruit. Its natural occurrence has been reported from India (Shanker *et al.*, 1972).

Transmission: The virus is sap transmissible and by *Myzus persicae* and *Sitohion rosaeformis* but not transmitted through seed.

Host range: The virus is restricted to family cucurbitaceae. *Cucurbita pepo*, *Cucumis melo*, *C. melo* var. *Utilissima*, *Lagenaria*

siceraria vars. round and long. *Luffa acutangulata*, *Citrullus vulgaris*, *Momordica charantia*, *Benincassa hispida* and *Trichosanthes anguina*, *Cucumis sativus* is a symptomless carrier.

Properties: The virus is inactivated at 50° C. at a dilution of 1:500 and in eight hours at room temperature (32–35° C) and in twenty-six hours at 8°C. The virus is successfully purified using 0.1M citric acid phosphate buffer (pH 8) containing 10 mg DIECA as an extracting fluid followed by three cycles of differential centrifugation and agar gel filtration. The purified virus preparation had maximum UV absorption peak at 260 μ and $S_{20}W$ value of 169 in an analytical ultra centrifuge. The electron microscopy revealed the virus particles to be long flexuous threads with modal length of 840 nm and an average width of 15 nm. Antiserum prepared was found to be specific and had a titre of 1:2,048 in precipitin tube tests. The pumpkin mosaic virus does not react with antisera of *Cucumis* viruses 1 and 2. It resembles the *Cucumis* virus 3 reported on vegetable marrow and bottlegourd mosaic virus and has close affinities with watermelon mosaic virus reported from other countries (Shankar *et al.*, 1972).

VEGETABLE MARROW (*Cucurbita pepo* L.)

Mosaic

In the field the virus affected plants show three main types of symptoms, namely, mosaic, filiform and witches broom (Reddy and Nariani, 1963).

Symptoms: The vegetable marrow plants infected with mosaic disease show typical mosaic patterns of light and deep green on the affected leaves and reduction in leaf size. Flowering is delayed, and fruits are reduced in size, paler in colour and occasionally chlorotic spots are seen on the outer skin of the young fruits.

In the case of filiform type severe distortion of leaf lamina and filiformy of leaves are the striking features. The younger leaves show vein clearing followed by chlorosis and the older leaves show prominent dark green blisters, the rest of the lamina being chlorotic. Vein and veinlets extend beyond the margin due to the reduction of interveinal tissue. In the advanced stage, the leaves are often reduced to veins and narrow strips of surrounding leaf tissue. Flowering is delayed and in severe cases the plant does not set normal fruits.

The witches broom type of symptoms are seen generally in the late stage of the crop and are characterised by a dense tuft of irregularly bent, stout and stunted branches producing severely reduced and malformed leaves. In early stages the branches produce numerous axillary buds pale green in colour, which develop very late into small leafy shoots. The petioles and internodes are very much reduced resulting in a witches broom like appearance. A few fruits develop and are small. The fruits that survive do not set seed or may set poorly developed seed.

Only the first two types have been studied in detail in India (Reddy and Nariani, 1963).

Transmission: The mosaic type is transmitted by *Aphis gossypii*, *A. craccivora*, *A. evonymi* and *Myzus persicae* and is also seed transmissible. The filiform type is transmitted by *M. persicae* only. Both are also readily sap transmissible. Vegetable marrow mosaic virus (VMMV) (*Cucumis virus 1*) is seed borne to the extent of 6.3 per cent and can be controlled by thermotherapy, i.e., by hot air treatment at 70°C for two days or at 40°C for four weeks (Sharma and Chohan, 1971).

Properties: In case of mosaic disease the virus is found to be infective when heated to 52°C for ten minutes, but not at 55°C for the same period. The dilution end-point of the virus ranges between 1:200 to 1:300. It resists ageing *in vitro* for twelve hours but not for sixteen hours at room temperature (32–35°C).

In the case of filiform type, the virus has its thermal inactivation point between 55–60°C for ten minutes. The virus survives twenty-four hours at 32–35°C and withstand nine days at 7°C.

Host range: In case of mosaic type the virus infects *Cucumis sativus* C. melo cv. Utilissima, *C. anguria*, *Cucurbita moschata*, *C. maxima*, *Lagenaria siceraria* cvs. Round and Long, *Trichosanthes anguina*, *Momordica charantia*, *Luffa acutangula*, *L. cylindrica*, *Citrullus vulgaris* cv. Fistulosus, *Zinnia elegans*, *Gomphrena globosa*, *Citrullus vulgaris*, *Brassica campestris* L. cv. Sarson and *Hesperis matronalis*, *Nicotiana tabacum*, *N. rustica* and *N. glutinosa*. The filiform type virus infects cucurbitaceous plants. The mosaic type virus resembles *Cucumis virus 1* whereas the filiform type is caused by *Cucumis virus 3* (Reddy and Nariani, 1963).

Yellow Mosaic

Symptoms: The infected plants stand out exhibiting a striking

yellow vein mosaic in leaves. The disease has been reported from India.

Transmission: It is not sap transmitted but only by *Bemisia tabaci* (Varma, 1955).

Host range: Includes *Cucurbita moschata*, *Luffa acutangula* and Dilpas and cv. *Citrullus fistula*.

Yellow Vein Mosaic of *Cucumis Sativus*

Cohen and Nitzany (1960) described a virus causing typical yellow vein mosaic in *Cucumis sativus* (cv. Beit Alfa) from Israel.

This virus differs from all other whitefly transmitted viruses described so far in being easily mechanically transmitted, in addition to its whitefly vector.

The overall symptom picture is strikingly similar to that described by Varma (1955) in *Cucurbita pepo* which is also transmitted by *Bemisia tabaci* and infects *Cucumis sativus* but is not sap transmissible.

TORI (*Luffa acutangula*)

Mosaic

Symptoms: The disease is characterised by light and dark green mosaic mottling, downward curling of leaf margins and general stunting in plant growth. Affected plants bear fewer flowers and fruits. The disease has been reported from India (Mitra and Nariani, 1965).

Transmission: The virus is found to be transmissible by mechanical sap inoculation but is neither transmitted through seed nor by *Myzus persicae*, *Aphis craccivora*, *Rhopalosiphum maidis*.

Properties: The virus has a thermal inactivation point of 55-60°C, dilution end-point ranges between 1:500 and 1:1000 and longevity *in vitro* of four to six hours at 38-41°C, six to eight hours at 8-10°C and twenty-four to forty-eight hours at 0°C. It is viable in desiccated leaves for three days but is inactivated after four days.

Host range: Host range of the virus is found to be restricted to family cucurbitaceae. The virus is transmitted to *Luffa cylindrica*, *Lagenaria siceraria*, *Cucurbita moschata*, *C. pepo*, *Citrullus vulgaris* cv. *fistulosus*, *Momordica charantia* and *Trichosanthes anguina*.

The causal virus is serologically unrelated to *Cucumis virus* 2C and is identified as *Cucumis virus* 3 (Mitra and Nariani, 1965).

WATERMELON (*Citruillus vulgaris*)**Mosaic**

Symptoms: The main symptoms are mild chlorosis, stunting, distortion and mottling. The mottling usually consists of green bands along the veins or of raised green blisters and wide chlorotic interveinal areas.

Transmission: The virus is mechanically sap transmissible but is not seed-borne. *Myzus persicae* and *Aphis gossypii* are the principal vectors but other aphids have also been reported.

Properties: The thermal inactivation point is 55 to 60°C, the dilution end-point is between 1:10,000 and 1:30,000 and the longevity *in vitro* nine to ten days at room temperature (Anderson, 1954). The virus particles are flexuous thread like 700 800 nm long (Van Regen Mortel, 1960).

Host range: Cet is restricted to family cucurbitaceae. It includes *Cucurbita pepo*, *C. maxima*, *C. moschata*, *Cucumis sativus*, *C. melo*, *Lagenaria ciceraria* and *Luffa* spp. The virus was first reported from Florida (Anderson, 1954). Later some strains have been reported from South Africa (Van Regen Mortel *et al.*, 1962) Venezuela (Lastra, 1968) and California (Milne and Grogan (1967). In India, the virus has been reported on *Cucurbita pepo* (Bhargava and Joshi, 1960; Reddy and Nariani, 1963) and pumpkin (Shankar *et al.*, 1972).

Vein Banding Mosaic

Symptoms: The chief symptoms of the disease are the diffuse mottling consisting of irregular light green areas on dark green background of the leaf. Frequently young leaves show vein banding symptoms. The old leaves show a more conspicuous mottling with dark green areas slightly raised in the form of blisters. The diseased vines set fewer and smaller fruits and are conspicuous because the vines of the runners protrude stiffly above the general level of the vines. The infected shoots show a shortening of internodes resulting in crowding of young leaves which appear somewhat stunted and rolled. It has been reported from India (Shankar and Nariani, 1974).

Transmission: The disease is sap transmissible. Transmission tests with several aphid species gave negative results. The virus is not seed borne.

Properties: The virus has a thermal inactivation point of 95–98°C, dilution end-point ranges 1:10,000 and 1:50,000 and longevity *in vitro* of more than a year. The purified preparation has a rigid rods with a modal length of 240 nm (Shankar and Nariani, 1974).
Host range: It is limited to cucurbitaceae but induces local lesions on *Chenopodium amaranticolor*. It infects, watermelon, cucumber, *Cucumis melo*, *C. anguria*, bottlegourd, *Cucurbita pepo*, *Luffa acutangula*, *Trichosanthes anguria*, and *Citrullus vulgaris* cv. *Fistulosus*. *Momordica charantia* is a symptomless carrier.

The casual virus is serologically related to *Cucumis virus 2*.

GUAR (*Cyamopsis tetragonoloba*)

Necrosis

Symptoms: Symptoms consist of necrotic tissue, varying considerably in diameter according to the environmental conditions, surrounded by infected but apparently healthy tissue on the entire leaf.

Transmission: The virus is transmitted mechanically.

Host range: The virus infects few varieties of bean, cowpea, petunia, datura and White Burley tobacco.

The virus has been identified as a strain of tobacco ring spot virus occurring in India (Verma *et al.*, 1963).

ONION (*Allium cepa*)

Yellow Dwarf Virus

Symptoms: The disease is identified by the presence of a series of short chlorotic streaks on the leaves. The chlorotic streaks eventually become yellow throughout. Later the leaves become crinkled and somewhat flat. Flower stalks of infected plants also show yellow streaks extending upward from the base. Later the streaks coalesce, the stalks become dwarfed, yellow and somewhat twisted in a characteristic manner and bear fewer flowers. The disease has been observed in India.

Transmission: The disease, could not be transmitted by sap inoculation, rubbing or by the pinpricking. The virus can be transmitted by injecting juice extracted from diseased onion bulbs and leaves into healthy bulbs. The disease is also transmitted by *Aphis gossypii*, *A. evonymi*, *A. craccivora*, *A. maidis*, *Myzus persicae*,

Longiunguis sacchari (Dhingra and Nariani, 1963).

Control: Use of virus free bulbs for planting is recommended.

GARLIC (*Allium sativum*)

Mosaic

Symptoms: A severe mosaic infection in garlic locally known as Bhutani Garlic, has been observed throughout Darjeeling Hills in India. The incidence has been observed upto 12 per cent in some severely affected belts.

Transmission: The virus is sap transmissible. The virus is also transmitted through *Myzus persicae* and *Aphis gossypii* but not by *Brevicoryne brassicae*.

Properties: The virus is infective at a dilution of 1:5000 but not at 1:10,000 and is completely inactivated at 67°+2°C and by storage at room temperature for eighty hours.

Host range: The virus could not be transmitted to *Allium cepa* and Narcissus varieties, the indicator varieties for onion yellow dwarf virus. The disease is reported to be infecting garlic only indicating its difference from onion yellow dwarf virus.

LETTUCE (*Lactuca sativa*)

Mosaic

Symptoms: The virus disease is identified by dwarfing, defective hearting, mottling and distortion of leaves. Clearing of veins is common and in addition there may be an irregular pale blotching on the whole leaf.

Transmission: The virus is sap transmissible and is also carried through seed upto two per cent. *Aphis gossypii*, *A. evonymi* and *Myzus persicae* have been shown to be the vectors of the virus (Nariani and Pathanian, 1960).

Properties: The virus has a thermal inactivation point of 50-60°C, dilution end-point ranges between 1:50 and 1:100 and longevity *in vitro* for twenty-four to forty-eight hours at 24°C.

Host range: The virus infects *Pisum sativum*, *Gomphrena globosa* and *Lathyrus odoratus*, which are symptomless carriers of the virus. The disease has been observed in India.

Yellow Mosaic

Symptoms: The virus is characterised by the presence of yellow patches on the leaves along with green colour forming mosaic.

Transmission: The virus is sap transmissible and is also carried through seed upto 30 per cent.

Properties: The virus can withstand heating at 86°C for ten minutes, a dilution of 1:66,000 and storage for sixty days at 15–25°C.

Host range: The virus infects tobacco cvs. Harrison's Special and White Burley and tomato cv. Sutton's but not sweet pea.

Yellow mosaic is considered to be distinct from other viruses affecting lettuce which is reported from India (Vasudeva *et al.*, 1948).

Necrotic Yellows

Symptoms: The disease is identified by fairly rapid transition from the normal healthy green to full green, bronzing or necrosis on some leaves, severe chlorosis, flattened growth, flaccidity and death of plants.

Transmission: The virus is transmissible by mechanical means. The vector of the disease is *Hyperomyzus lactucae*. The virus is of circulative persistent type.

Properties: The thermal inactivation point is 52–54°C, the dilution end-point is 10^{-2} and the longevity *in vitro* is one to eight hours (Stubbs and Grogan, 1963). The particle is bacilliform or bullet shaped 66 nm wide and measuring about 227 nm in length. The particle has an outer membrane (Harrison and Crowley, 1965; Wolanski *et al.*, 1957). The virus probably contains RNA and the fact that infectivity is destroyed by chloroform and diethylether suggests that it contains lipid (Matthews, 1970).

Host range: The virus infects *Lactuca sativa*, *Scmola*, prickly lettuce, *Sonchus*. The disease is reported from Australia, New South Wales and Brazil.

Control: Eradication of the sow-thistle, which is the chief source of virus.

PEA (*Pisum sativum*)**Mosaic**

Symptoms: The diseased plants are pale, weak and dwarfed. The young leaves and stipules show a general mosaic mottling

whereas the older ones display distinct irregular whitish areas. Tendrils are curled abnormally and the apical leaves remain folded. The diseased plants flower later than the healthy ones and flowers are smaller in size and fewer in number.

Transmission: The virus is sap transmissible and is also transmitted by *Aphis craccivora*, *A. gossypii*, *A. evonymii*, *Myzus persicae*, *Rhopalosiphum pseudobrassicae* and *R. maidis* but not through the seed of infected pea, *Vicia faba* or *Trigonella foenum-graecum* (Sreenivasan and Nariani, 1966).

Properties: The virus has a thermal inactivation point of 58–60°C and a dilution end-point between 1:1000–1:2000. The longevity *in vitro* of the virus is found to be twenty-four to forty-two hours at room temperature (19–27°C), sixty-six to seventy-two hours at 14–16°C and seventy-two to ninety hours at 8–10°C. It has an optimum 'pH' infection range from six to eight.

Host range: The virus is transmitted to *Pisum sativum* var. Arvense, *Vicia faba* L., *V. narbonensis*, *V. sativa*, *V. biennis*, *Lathyrus odoratus* L., *L. ochrus*, *L. sativus*, *Trigonella foenum-graecum*, *T. corniculata*, *Crotalaria juncea*, *Lupinus albus*, *augusifolius* and *Cicer arietinum*. *Chenopodium amaranticolor* produces countable chlorotic local lesions on the inoculated leaves (Sreenivasan and Nariani, 1966).

Varietal resistance: Pea varieties Cannors perfection, Horal, Hundredfold and Little Marvel are immune to infection. Besides India this disease is reported from Japan, Australia, USA and British Islands.

PHASEOLUS LONGEPEDUNCULATUS

Yellow Mosaic

Symptoms: This disease induces angular, chlorotic spots on the leaflets. The disease has been reported from Brazil (Flores and Silberschmidt 1966).

Transmission: The virus is transmitted by *Bemisia tabaci*.

It is regarded as a strain of Abutilon mosaic virus (Flores and Silberschmidt, 1966).

Further information is lacking.

POTATO (*Solanum tuberosum*)**Aucuba Mosaic (Potato Virus F or G)**

Symptoms: The disease is characterised by bright-yellow spots on lower leaves. Some varieties show a brilliant and extensive yellow spotting and necrotic tubers.

Transmission: The disease is sap transmissible and also by *Myzus persicae*. The losses caused by virus aucuba mosaic vary from 20 to 40 per cent.

Properties: The thermal inactivation point is 65°C, dilution end point is between 1:200 and 1:500, and the longevity *in vitro* is less than four days at 15°C. The virus particle is a long rod shaped about 480 to 580 nm in length.

Host range: It infects *Datura stramonium*, tobacco, Petunia, *Solanum nodiflorum* and *Capsicum annuum* (Kassanis, 1961).

Calico Virus (Alfalfa Mosaic Virus)

Occurrence of this virus in potato in India was first reported by Nagaich and Giri (1968). One group of strains cause yellowing of leaves in patches, whereas the symptoms caused by other type of strains are more or less like that of potato virus Y.

In commercial potato varieties the losses vary from 15 to 40 per cent.

This virus is sap transmissible as well as by *Myzus persicae* and *Aphis rumicis* (*A. evonymii*).

Foliar Necrosis

Symptoms: On Phulva variety it produces very mild mottling without any distortion of the leaf followed by faint chlorosis. Then necrotic spots appear which finally coalesce and form larger necrotic areas (Ganguly and Raychaudhuri, 1964).

Transmission: The virus is transmitted by mechanical inoculation and also by grafting.

Properties: The dilution end-point of the virus ranges between 1:5,000 and 1:10,000. It can withstand exposure to 60°C for ten minutes but is rendered completely innocuous when exposed to 68°C for the same period. The virus is completely inactivated in seven to eight days when stored at 25–30°C. It loses infectivity after nineteen to twenty-one days at 0–2°C. It remains infective even after thirty days, but is rendered completely innocuous after

storage for thirty-nine to forty-two days. It is found that pH infectivity of the virus ranges between two to nine and 7.4.

Host range: The virus infects the many solanaceous hosts like *Solanum tuberosum* cvs. Phulwa, President and Up-to-date, *S. nodiflorum*, *N. tabacum*, *Datura stramonium*, *Lycopersicon esculentum* and *Capsicum annum*. Disease has been observed in India.

Control: Potato seed certification.

Leaf Curl

Symptoms: Symptoms are identified by reduced size of rough leaves margins curling up and rough surface. The disease has been observed in Venezuela. (Wolf *et al.*, 1949).

Transmission: The virus is transmitted through *Bemisia tuberculata* and *Alcurotrachelus socialis*.

This disease is probably a complex involving at least three distinct viruses, namely, tobacco leaf roll, cotton leaf curl and cassava leaf curl (Wolf *et al.*, 1949).

Leaf Roll

Symptoms: The infected potato plants have thickened leathery leaves which also develop pronounced rolling. The young infected leaves are pale in colour on the upper side and pinkish on the lower side. The infected plants are stunted and leaves tuber early. The whole plant is very harsh to touch and rattles when shaken. Tubers grown on such plants show a network of necrosis which is confined to the phloem strands. The disease was observed in India in 1943 (Pal, 1943).

Transmission: The virus is not sap-transmissible but is transmitted by the green-peach aphid, *Myzus persicae*.

Properties: The virus is inactivated by treatment of tubers of several potato varieties at a constant temperature of 35°C for eight weeks or more, these showed differences in tolerance to heat (Nagaich and Upreti, 1964). Hot water treatment is also applied for the inactivation of leaf roll virus and is a quick method (Upreti and Nagaich, 1968). The virus is inactivated when whole tubers were treated for seventeen minutes at 55°C. Cut tubers treated for twenty-five minutes at 52°C could be freed from leaf roll virus.

Host range: It infects *Lycopersicon esculentum*, *Datura stramonium*, *D. tatula*, *Physalis angulata* and *P. floridana*.

- Control:** (i) By roguing of the diseased plants,
(ii) Planting certified healthy seeds.
(iii) Inactivation of the virus in tubers and then planting them (Nagaich and Upreti, 1964 and Upreti and Nagaich, 1968) and
(iv) applying granular systemic insecticides at the time of planting and earthing up (Mukhtiar Singh *et al.*, 1970).

Mild Mosaic (Potato Virus X)

Symptoms: The disease is characterised by the loss of chlorophyll, that is the presence of alternating patches of light and dark-green colour on the leaf. In severe cases leaves become puckered and small. Occasionally the plants become stunted (Vasudeva and Lal, 1944).

The symptoms differ widely, depending upon the potato variety and the strain of the virus involved. The disease has been commonly observed in India.

Transmission: The virus is sap-transmissible. No insect vector is known although the virus has been shown to be transmitted by grass hopper *Melanoplus differentialis* (Walters, 1952).

A severe strain of ring spot virus of potato virus X of 'Gola' variety has been reported from India (Sharma and Raychaudhuri, 1962).

Properties: Thermal inactivation point of the virus is 70°C. Dilution end-point ranges between 1:10⁵ to 1:10⁶. Longevity *in vitro* varies from several weeks to a year at room temperature. Virus particles are long flexuous rod measuring about 515 nm in length.

Host range: It infects *Nicotiana tabacum*, *N. glutinosa*, *Datura stramonium*, *Petunia* sp., *Lycopersicon esculentum*, *Solanum nigrum*, *S. melongena*, *Capsicum frutescens*, *Gomphrena globosa* and *Chenopodium amaranticolor* and produces local lesions.

Control: Seed certification.

Rugose Mosaic (Potato Virus X and Y)

Symptoms: The symptoms consist of dwarfing of plants with tubers reduced in size. The lower leaves generally have black necrotic veins, while the upper leaves are mottled light green spots. The spots are abundant near leaf ribs on the younger leaves. The foliage is wrinkled or ruffled.

This disease is a composite virus disease caused, by the

combination of two viruses—Potato virus X and Y. In India the disease is commonly found in potato variety Phulwa.

Severe Mosaic (Potato Virus Y)

Symptoms: Infected plants are characterised by a blotchy mottle on the underside of younger leaves. The leaves become completely necrotic and withered, but remain hanging as if attached to the stem by a thread. The topmost leaves are not necrotic but frequently mottled and slightly crinkled. Plants raised from infected tubers show little necrosis or leaf drop streak, but are small and stunted and their leaves and stems are very brittle. The internodes are short and the leaves are generally mottled, twisted and bunched together (Pal, 1943). The disease is very common in India.

Transmission: The virus is transmitted readily by sap inoculation by aphids, the most important and efficient being *Myzus persicae*. The virus, however, is not carried in true seed of potato.

Properties: The thermal inactivation point of the virus ranges between 52–55°C dilution end-point ranges between 1:100 to 1:1000. Longevity *in vitro* is twenty-four to forty-eight hours. Virus particles are long and flexuous measuring 700 nm in length.

Host range: The virus can infect tobacco producing the characteristic vein banding symptoms. It also produces local lesions in several plant species. Of these *Physalis floridana* and *Lycium rhombifolium* are the most satisfactory differential hosts. *Datura stramonium* is immune to infection with potato virus Y (Smith, 1932).

Control: The disease can be controlled by the following three methods.

- (i) Use of disease free certified seed potato.
- (ii) Inspection of the crop for detecting and roguing of diseased plants in the early stages of crop growth.
- (iii) Spraying with *Metasystox* occasionally against the aphid populations.

Super Mild Mosaic (Potato Virus A)

Symptoms: The disease is characterised by yellowing of the leaf margins and yellow mottle on the entire lamina with crinkling of the leaves (Vasudeva and Ramamurthy, 1946). Generally the virus induces faint mottling on the leaves without any deformation, and the plants are not much stunted. The disease has been observed in India.

Transmission: Virus is sap transmitted. Insect vectors are *Myzus persicae*, *Aphis gossypii* and possibly *M. circumflexus*.

Properties: The virus is inactivated by heating at 50°C for ten minutes. Dilution end-point ranges between 1:50 to 1:100. Longevity *in vitro* ranges between 12 to 24 hours.

Control: Potato seed certification.

Yellow Vein Virus

Symptoms: The chief symptom of the disease is the bright yellow colouration in the leaf veins but the colour fades away as the plants grow old.

Transmission: The virus is transmitted by white fly (Smith, 1957). The disease may be the same which is called vein yellowing and has been mentioned by Silberschmidt (1954) from Southern Columbia and by Alba (1950) from Ecuador.

RADISH (*Raphanus sativus*)

Mosaic

Symptoms: The symptoms are mosaic mottling of young leaves which is often associated with circular interveinal chlorotic areas which gradually increase in size and finally coalesce to form irregular patches. The affected plants are stunted and their leaves reduced in size. Frequently necrotic lesions appear on the stem, petiole and midrib of the affected plants. When necrotic lesions appear on the midrib leaf first bends outwards and then the midrib breaks at the necrotic region so that a part of the leaf collapses.

Transmission: The virus is transmitted by mechanical inoculation (Raychaudhuri and Pathanian, 1955).

Properties: The thermal inactivation point of the virus ranges between 85–90°C, the dilution end-point is 1:10,00,000 and ageing at room temperature (17–22°C) is seventeen days but at 6 to 8°C the virus retains infectivity even after storage for 101 days.

Host range: It is restricted to cruciferae and the important hosts apart from radish are *Brassica rapa*, *B. campestris*, *B. nigra* and *B. alba*.

The disease has been reported from India.

SWEET POTATO (*Ipomoea batatas*)**Mosaic Virus A**

Symptoms: The virus is carried without symptoms in some varieties of sweet potatoes; in others it produces large number of chlorotic spots on all leaves and only rarely causes mosaic symptoms.

Transmission: The virus is not mechanically transmitted but is transmitted by the vector *Myzias persicae*.

Host range: It is transmitted to other species of *Ipomoea* by grafting (Sheffield, 1957, 1958). The disease has been reported from East Africa.

Vein Clearing (Mosaic Virus B)

Symptoms: The symptoms on sweet potato are stunting, with shortening of the internodes, reduction in leaf size and general chlorosis. Leaves may be leathery in consistency. Yellow mottling is more clear in broad leaved than in the narrow-leaved varieties (Sheffield, 1957, 1958). The disease is also characterised by pronounced clearing of the veins (Loebenstein and Harpaz, 1960). Most varieties develop clearing of the midrib and primary veins on the expanding leaves followed by a discontinuous banding of primary, secondary and smaller veins with light green to yellowish areas of tissue (Robertson, 1964).

Transmission: The virus cannot be transmitted mechanically to sweet potato, but can be transmitted by the vector *Bemisia tabaci*.

Host range: Virus infects other species of *Ipomoea* and several Solanaceous species including *Datura* sp., *Lycopersicon esculentum*, *Nicotiana glutinosa*, *N. tabacum*, *Petunia* sp. and *Physalis peruviana*.

The disease has been reported from East and South Africa and Israel.

TOMATO (*Lycopersicon esculentum*)**Aucuba Mosaic**

Symptoms: The disease is characterised by interveinal mottling and yellowing of the leaves with scattered patches on green areas. The leaf surface appears to be crinkled and brittle (Das and Raychaudhuri, 1953). The disease has been observed in India.

Transmission: The virus is readily transmitted by sap inoculation.

Properties: The virus can withstand temperature of 98°C and dilution of 1:100,000. Longevity *in vitro* is more than 77 days and the virus retains its infectivity after treatment with 90 per cent alcohol, but is inactivated by 95 per cent alcohol. The virus remains active even after 105 days in dried tissues at 28–30°C (Das and Raychaudhuri, 1953).

Host range: The virus infects *Solanum nigrum*, *S. melongona*, *N. rustica*, *Datura stramonium* and *Petunia* sp. (Das and Raychaudhuri, 1953).

Black Ringspot

Occurrence of the tomato black ring spot in India is described by Sastry (1966).

Symptoms: The virus disease is characterised in the beginning by the presence of small semi necrotic rings gradually becoming completely necrotic. In later stages several rings coalesce and give an appearance of big necrotic patches.

Transmission: The virus is transmitted mechanically. *Aphis craccivora*, *A. evonymi*, *A. gossypii*, *Lipaphis erysimi*, *Myzus persicae* and *Bemisia tabaci* were unable to transmit the virus.

Properties: The virus has a thermal inactivation point of 58–62°C for ten minutes exposure. Virus remains infective at 1:100 dilution but is rendered non-infective at 1:1000. The infective sap stored at 35–37°C loses its infectivity after three days. Sap stored at 0°C gave infection even upto twenty days. The virus is isometric and measures about 30 nm in diameter (Hollings, 1965).

Host range: The virus infects the following plants and produces symptoms on them, namely, *N. tabacum* cvs. White Burley and Harrison's Special, *N. glutinosa*, *N. paniculata*, *Petunia* hybrids, *Cucumis sativus*, *Cucurita pepo*, *Datura stramonium*, *Beta vulgaris*, *Spinacea oleracea*, *Tetragonia expansa* *Phaseolus vulgaris* *Tropeolum majus* and *Hyoscyamus niger* (Sastry, 1966).

Bunchy Top

Symptoms: The growth of infected plant is very much retarded with the result that they are markedly stunted. All leaves are reduced both in width and length, stand erect and bunched together at the apex of the plant to form a rosette. The disease has been reported from South Africa (McClellan, 1948).

Transmission: The virus is transmitted by mechanical inoculation and by grafting.

Properties: Thermal inactivation point is 60 to 70°C and longevity is twelve to twenty-four hours at room temperature.

Host range: The virus infects following additional hosts: *Datura stramonium*, *Nicotiana glutinosa*, *Physalis angulata*, *Solanum giganteronora*, *S. indicum* and *S. sisymbirifolium* (McClean, 1948).

There is no evidence that bunchy top disease is a complex, produced by more than one virus (McClean, 1948).

Condensed Top Disease

Symptoms: The disease is characterised by extremely shortened internodes and cessation of apical growth. The leaves are reduced in size and sometimes folded upwards, and are dark-green. There is leaf and stem necrosis and flower shedding also occurs (Yassin and Nour, 1965). The disease has been reported from Sudan.

Transmission: The disease is graft transmissible. No transmission was achieved by *Bemisia tabaci* and unidentified capsid (Tassin and Nour, 1965).

Leaf Curl

Symptoms: The affected plants are stunted and the leaves and internodes are greatly reduced in size. The leaves are curled and crowded together. The leaflets are deformed and their margins are curled inwards or outwards. Infected plants are pale and tend to produce stunted lateral branches, which results in bushy growth of the plant. The virus causes partial or complete sterility. In case of early infection, no fruit formation takes place and only deformed fruits are formed.

Leaf curl virus disease is the most common one on tomato in India (Vasudeva and Samraj, 1948) and in Sudan (Yassin and Nour, 1965).

Transmission: The disease is transmitted by *Bemisia tabaci* and by grafting but not by mechanical inoculation (Vasudeva and Samraj, 1948).

Host range: All the tomato varieties tested against this virus proved to be susceptible, but *Lycopersicum peruvianum* showed high degree of resistance (Nariani and Vasudeva, 1963). The causal virus is the tobacco leaf curl virus which infects tobacco, chilli, datura, sunn hemp and various other weeds and ornamental plants.

Control: Dipping of seedling roots in gibberellic acid or 2-thiouracil (50 ppm) delayed the appearance of symptoms and reduced the severity of the disease whilst increasing the yield (Mukherjee and Raychaudhuri, 1966).

Enation Leaf Curl

Symptoms: The disease is characterised by curling, twisting and rolling of the leaves and dark green vein enations on the under-surface of the leaflets (Nariani, 1968).

Transmission: The disease is transmitted by grafting and also by *Bemisia tabaci* (Nariani, 1968).

Host range: The virus infects tobacco, *Datura stramonium* and *Capsicum annuum* (Nariani, 1968).

It is considered to be a strain of tobacco leaf curl virus and is designated *Nicotiana* virus 10 A or tomato enation leaf curl virus (Nariani, 1968).

Spotted Wilt

Symptoms: The first sign of the disease is bronze colour markings on the upper side of young leaflets which may be accompanied by a slight downward curling of the leaves. These may extend to petiole, stem, pedicel and calyx. Upward marginal rolling and stiffening of leaflets follow and small circular necrotic spots commonly occur on the leaves under favourable conditions. Necrosis extends to the stem near the growing tip, causing the latter to wilt and die. Sometimes mottle, chlorotic spotting and malformation may occur on leaves. On the unripened fruits yellow spots appear, often with distinct concentric zones of shades as yellow or bronze alternating with green and later with pink or red (Bald and Samuel, 1931). The disease has been reported from Australia.

Transmission: The virus is transmitted through the insect vectors which include *Thrips tabaci*, *Frankliniella insularis*, *F. occidentalis*, *F. moultoni* (Best 1937).

Properties: The virus is inactivated by heating at 42 to 45°C for ten minutes, at dilution of 1:10,000 and by ageing at 20°C for five hours. The virus is spherical in shape about 70 nm in diameter (Le, 1964; Black *et al.*, 1963). Some particles have tails suggestive of those of bacteriophages (Van Kammen *et al.*, 1966).

Control: The disease is controlled to some extent by elimination of overwintering hosts and also by control of thrips.

Curly Top

Symptoms: Symptoms consists of vein clearing or a slight yellowing along some small veins of partly developed leaflets. They then roll upwards and their veins become more noticeable than the veins of normal leaflets. Infected plants become more stunted.

Transmission: The virus is transmitted by leafhopper, *Agalia albidula* and by *A. ensigera*.

Host range: The host range of the virus includes Jimson weed and *Solanum nigrum*. (Costa, 1952).

Studies on curly top of tomatoes (*Lycopersicum esculentum*) and tobacco (*N. tabacum*) in Brazil, afforded evidence that these diseases are caused by a strain of the sugarbeet curly top.

TURNIP (*Brassica rapa*.)**Crinkle Virus**

Symptoms: The virus causes crinkling, stunting and rosetting of plants. The disease has been observed in India (Verma and Varma, 1961).

Transmission: The disease is transmitted mechanically but not by aphids.

Properties: Its dilution end-point is 10^5 and thermal inactivation point lies between 85–90°C.

Host range: It infects cauliflower, kohlrabi, cabbage, tobacco, tomato, potato, *Solanum nigrum*, *Datura stramonium*, *Cynopsis tetragonoloba* and sugarbeet (Verma and Varma, 1961).

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23

Weeds

ABUTILON SP.

Mosaic

Symptoms: The virus causes yellow and green patches of the leaves (Baur, 1906).

Transmission: The virus is not sap inoculable and can be transmitted by grafting and seed (Keur, 1933). The virus has whitefly *Bemisia tabaci* as its insect vector (Orlando and Silberschmidt, 1946). It occurs in Brazil, Puerto Rico, India and probably Trinidad.

Host range: The virus infects *Abutilon thompsonii*, *A. mulleri* and *A. megapotamicum* variegatum. In addition to these it infects *A. striatum* var. *Spurium* and *Sida rhombilolia* (Orlando and Silberschmidt, 1946). Costa (1955) was able to transmit the virus to cotton, okra, field beans, soybean, peanut, lupine, lima bean, lentil and other leguminous plants. It also infects potato and *Nicandra physaloides*.

ACALYPHA INDICA.

Yellow Mosaic

Symptoms: The virus causes mottling and chlorosis in affected leaves. In severe cases, reduction in size and puckering of the leaves are noticed. Chenulu and Pathak (1965) reported it from India.

Transmission: The virus is transmitted by the whitefly *Bemisia tabaci*.

Host range: The virus is restricted only to *Acalypha* sp.

ACHYRANTHES ASPERA**Mosaic**

Symptoms: The disease is characterised by dark green mosaic mottling of the leaves. In severely infected plants, the leaf size is somewhat reduced and the affected leaves at times show puckering.

Transmission: The virus can be easily transmitted by *Aphis gossypii*. Attempts to transmit the disease by *A. craccivora* were not successful. The virus is not sap transmissible.

Host range: The virus is restricted to the genus *Achyranthes* (Verma and Singh, 1972) and has been reported from India.

AGERATUM CONYZOIDES**Yellow Vein Mosaic**

Symptoms: The leaves of diseased plants exhibit a typical yellow vein mosaic with slight thickening of the veins on the underside of leaves (Cadd and Loos, 1941). Affected plants are considerably reduced in height but flowers and fruits are normal.

Transmission: The virus cannot be transmitted through sap or through seeds. It is only transmitted by *Bemisia tabaci* or through grafts.

Host range: The virus infects *Browallia elata* and *Vernonia* sp. On the former host a typical curl results, while on *Vernonia*, it gives rise to typical yellow vein mosaic symptoms.

BERMUDA GRASS (*Cynodon dactylon*)**Mosaic**

Symptoms: The diseased leaves show light green to yellow streaks in the beginning. In later stages more severely affected leaves are found to be chlorotic and sometimes with broken streaks (Bhargava *et al.*, 1971). The disease has been reported from India.

Transmission: The virus is readily transmissible by mechanical means and by *Aphis nerii*, *A. gossypii*, *Myzus persicae* and *Rhopalosiphum maidis*.

Properties: The infective sap is inactivated *in vitro* between 50–55°C and by dilution in between 1:100 to 1:500. It loses infectivity after being stored for three hours at room temperature.

Electron micrographs showed that virus particles range from 509 nm to 632 nm, with most of the particles being 507 nm. Cross protection and serological tests showed that the virus is distinct from maize mosaic and sugarcane mosaic viruses.

Host range: The virus is transmitted to maize, sorghum and *Pennisetum pedicellatum* (Bhargava *et al.*, 1971).

BOERHAAVIA DIFFUSA

Mosaic

Symptoms: The disease is characterised by dark green mosaic mottling of the leaves. In severe infection, the leaf size is somewhat reduced and the affected leaves sometimes show slight puckering. Singh and Verma (1972) reported it from India.

Transmission: The disease is readily grafted transmissible. Attempts to transmit the disease by the aphids (*Aphis gossypii*, *A. craccivora* and *Myzus persicae*) and by sap were unsuccessful. The virus could not be transmitted by whitefly (*Bemisia tabaci*).

Host range: The host range of the virus is restricted to the genus *Boerhaavia* (Singh and Verma, 1972).

EUPHORBIA PRUNIFOLIA

Mosaic

Symptoms: The diseased plants show very striking leaf mottle and chlorosis of leaf veins (Costa and Bennett, 1950). Infected leaves produce chlorotic areas of irregular shape on the leaf lamina with wrinkling or puckering of the leaf surface. The disease is reported from Brazil.

Transmission: Transmission percentage by sap is very low to *Euphorbia prunifolia* and *Datura stramonium*.

Bemisia tabaci transmits the virus to both hosts but not to thirty-six other hosts tested. No seed transmission was noted. The virus is carried in a persistent manner by the insect vector.

Host range: *Datura stramonium*, *Egophyllum esculentum*, *Nicondra physaloides*, *Oxalis* sp. and *Phyllanthus corradonis* (Costa and Bennett 1950).

HENBANE (*Hyoscyamus niger*)**Mosaic**

Symptoms: The disease is characterised by the development of vein banding. Infected plants become slightly stunted and after several weeks exhibit typical yellow mosaic mottle. Chenulu *et al.* (1968) reported it from India.

Transmission: The virus is sap-transmissible with or without any abrasive.

Properties: The virus has a dilution end-point between 1×10^{-6} and 10^{-8} . The thermal death point of the virus lies between 95–98°C. The longevity *in vitro* of the virus is crude juice at room temperature (24–30°C) is found to be fourteen days. The virus retains infectivity after continuous freezing for forty-five days. The electron micrographs of the virus revealed flexuous rods measuring 270–750 nm in length.

Serological studies have shown that the virus is not related to TMV and Potato virus S (PVS) Chenulu *et al.*, 1968).

Host range: It infects *Comphrena globosa*, *Chenopodium* spp. *Capsicum frutescence*, *Datura metel*, *D. inoxia*, *D. stramonium*, *Lycopersicon esculentum*, *Nicotiana glutinosa*, *N. longiflora*, *N. peniculata*, *N. rustica*, *N. tubacum*, *Physalis peruviana*, *Petunia* hybrids, *Solanum nigrum*, *S. tuberosum*.

MALVASTRUM COROMANDELIANUM**Yellow Vein Mosaic**

Symptoms: The virus caused typical yellow vein mosaic symptoms. The disease is widely spread in India. (Rao and Varma, 1961).

Transmission: The disease is not sap transmissible but can be easily transmitted by cleft grafting as well as by the whitefly, *Bemisia tabaci*.

Host range: The virus has a wide host range and the symptoms of the disease in various hosts vary from vein clearing to complete chlorosis, with or without vein swelling and enations.

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Diseases Attributed to Mycoplasma and Rickettsia-like Organisms

Attention of the plant pathologists towards mycoplasmas or rickettsia-like organisms was brought for the first time only in 1967 by Prof. H. Asuyama and his co-workers. Based on the evidences: (a) electron microscopy (Doi *et al.*, 1967) and (b) tetracycline therapy (Ishii *et al.*, 1967) these Japanese group of workers suspected that mycoplasma or chlamydia-like organisms may be the causal agent for mulberry dwarf, potato witches' broom, aster yellows or paulownia witches' broom. All these diseases comprise a group called 'yellows' diseases which behave like aster yellows disease and till recently considered to be leafhopper transmitted, heat and tetracycline curable viral disease (Kunkel, 1951; Black, 1953; Maramorosch, 1953), but nobody could purify or get the electron micrograph of the virus particle. After the reports of Asuyama group, now a number of workers have demonstrated the association of mycoplasma-like bodies or organisms (MLB or MLO) with aster yellows as well as with a number of other yellows diseases characterised by proliferation of axillary buds, smalling of leaves, abnormalities in floral parts, etc. Some of these diseases are also suppressed by tetracycline group of antibiotics. Many good reviews are available on the subject (Whitcomb and Davis, 1970; Maramorosch *et al.*, 1970; Davis and Whitcomb, 1972; Ghosh and Raychaudhuri, 1972, 1974; Hampton, 1972). More recently association of Rickettsia-like organisms have also been suspected with a limited number of yellows diseases which will be dealt at the end of this chapter. Before describing the individual diseases, a brief account of mycoplasmas is essential for understanding their presumed equivalents in plants.

For many years, mycoplasmas were known as pleuropneu-

monia-like organisms (PPLO) and there was a lot of confusion about their taxonomic position. Though PPLO was first cultured in 1898 (Nocard *et al.*, 1898) the genus *Mycoplasma* was recognised only in 1956 (Edward and Freundt, 1956). Now it is an universally established and accepted genus of the order Mycoplasmatales under the class Mollicutes (soft skin). Due to their fastidious and confusing nature, the Subcommittee on the Taxonomy of Mycoplasmatales (1972) proposed some minimal standards for the description of a new species under Mycoplasmatales.

Mycoplasmal cells are the smallest known cells, lacking cell wall but are limited by a unit layer of plasma membrane (Table 1). The cells are highly pleomorphic, from coccoid to filamentous, capable of growing in cell-free medium and form typical minute 'fried-egg' shaped colonies. Ultrastructure of a typical mycoplasmal cell consists of a trilaminar membrane, surrounding cytoplasm which is packed with ribosomes and fibrillar deoxyribonucleic acid (DNA). The cell consists of a naked circular chromosome about 1000×10^6 daltons which replicate like the chromosome of *Escherichia coli*. Base ratio determination of the organisms have given some interesting data, in fact, recently with the development of sophisticated techniques and instruments; genome size, base ratio and DNA-hybridisation techniques present the strongest evidences for distinction between mycoplasmas and the related organisms: L-form of bacteria, Rickettsia and Chlamydiae.

Role of Mycoplasmas in Yellow Diseases

Under electron microscope, organisms associated with yellow disorders and their insect vectors show similar morphology, as the animal mycoplasmas. Electron microscopy of thin sections of diseased plant and vector tissues show pleomorphic cell wall-less bodies. These bodies are surrounded by triple-layered unit membrane and are usually filled with ribosome-like granular particles and fibrillar nuclear material.

American scientists working with corn stunt disease observed some helical filamentous structures in the cells of the infected plants under phase contrast and stereo-electron microscope (Davis *et al.*, 1972a, b; Steere and Davis, 1972). These filamentous bodies are helically coiled (0.2 to 0.25μ by 3 to 15μ) and are without cell wall but surrounded by a unit membrane. Though no locomotary

organs have been seen attached with them, they show flexing, bending and curling movement like *Spirochaetes*. Davis and Worley (1973) proposed the name '*Spiroplasma*' (spiral form) for the corn stunt organism instead of 'MLO'.

Attempts have been made to grow yellows agents (MLO) in cell-free media with a few success only. The cultures obtained by Saglio *et al.* (1971) from citrus stubborn has been characterised by the experts of mycoplasmas (Jole *et al.*, 1973; Saglio *et al.*, 1972) and deposited in American Type Culture Collection: ATCC 27556 (Morocco strain) and ATCC 27665 (California strain). Till further characterisation, the organism has been proposed as a new genus *Spiroplasma citri*, gen. nov. sp. nov. under 'Genera of uncertain affiliation' Mollicutes (Saglio *et al.*, 1973).

Apple Rubbery Wood

Symptoms: The disease is identified by the rubbery texture and flexibility of stem and branches of the tree. Fruiting trees develop a pendulous habit. Usually flexible condition of wood is attributed to incomplete lignification of xylem vessels and fibre cells. Generally, the affected trees are less vigorous than normal ones (Bhargava and Bisht, 1961; Dhingra and Raychaudhuri, 1970).

Transmission: The disease is found to be transmitted only by grafting and not by sap inoculation.

Electron microscopy: Electron microscopy of thin sections of the phloem cells of apple tree with rubbery wood disease revealed the presence of MLO (Beakbane *et al.*, 1971). These bodies were frequently filled with ribosomes and surrounded by a unit membrane.

Control: The disease causing agent is found to be uniformly distributed in plant cells and can be easily inactivated by heat by maintaining the plants at 36°C (Posnette *et al.*, 1962).

Brinjal (Egg Plant) Little Leaf

This disease was first reported from Coimbatore, South India (Thomas and Kishnaswami, 1939). Through the disease is common in South India, it is prevalent throughout the brinjal growing areas of India. Under natural conditions the disease incidence sometimes goes upto 80 to 90 per cent.

Symptoms: Detailed account on the symptomatology was given by Anjaneyulu and Ramakrishnan (1972a). Early symptoms

start with chlorosis in the young leaves. The flower buds take upright position and several axillary buds start sprouting. In advanced stage, the diseased plants show stunting due to extreme reduction in the size of the internodes and the leaves, and start producing phylloid flowers. Fruit setting rarely takes place in the affected plants.

Transmission: The disease is graft transmissible and in nature spread by the cicadellid vector, *Hishimonas phycitis*.

Electron microscopy: Spherical to avoid bodies 40–300 nm in diameter resembling mycoplasmas were shown to be associated with the phloem cells of the little leaf affected brinjal plants (Varma *et al.*, 1969, 1973).

Control: Preliminary tetracycline therapy of this disease was first reported by Anjaneyulu and Ramakrishnan (1969). Of the several chemicals screened under glasshouse and field conditions, tetracycline antibiotics, chloramphenicol (chloromycetin) and nitrofurazone were found to have temporary therapeutic effect (Raychaudhuri *et al.*, 1970; Anjaneyulu and Ramakrishnan, 1972b).

Citrus Greening

Greening disease of citrus is of major importance in India and South Africa. The disease has been found to be one of the major diseases of citrus distributed throughout India (Nariani *et al.*, 1967). Martinez and Wallace (1967) recorded the disease from the Philippines.

Symptoms: Symptoms may be produced in the whole tree or in few isolated branches. The chief symptoms of the disease are yellowing of midrib and lateral veins of the old mature leaves. Interveinal areas along the vein show yellowing, ultimately the whole leaf may sometimes turn yellow and shed with the onset of summer. This is followed by secondary growth from the axillary buds which consists of short, upright, small and weak shoots which may be bushy. These weak shoots show various types of discolourations on the leaves frequently resembling those caused by zinc or iron deficiency. In late infection, shoots may bear excessive premature flowers. Fruits produced on the affected trees are generally of poor quality and show conspicuous blotching (yellow patch) on the mature fruits on the side exposed to sun (McClean and Oberholzer, 1965a; Fraser *et al.*, 1966).

Transmission: Greening is graft-transmissible and has been transmitted by psyllid vectors *Trioza erythrae* in South Africa (McClellan and Oberholzer, 1965b). Another psyllid, *Diaphorina citri* has been responsible for its spread in India (Capoor *et al.*, 1967) and Philippines (Martinez and Wallace, 1967). Even a single psylla is capable of transmitting the disease and there is evidence of a latent period of about eight to twelve days in the vector (Raychaudhuri *et al.*, 1969; Nariani and Singh, 1971). The psylla is not able to transmit the greening disease during the span of its nymphal life but it can pick up the pathogen in nymphal stage and transmit when it is adult. Once infective, it can transmit the greening disease after a minimum infection feeding of four hours. It continues to spread the disease during its life period.

Electron microscopy: MLB have been found associated with the greening disease (Lafleche and Bove, 1970a).

Culturing of greening agent: MLO was isolated from greening affected plants and cultivated on cell-free media (Ghosh *et al.*, 1971, 1973). The medium used was PPIO agar or PPIO broth (Difco Laboratories, USA) supplemented with 20 per cent serum. In preliminary experiments greening symptoms could be induced using this culture (Raychaudhuri *et al.*, 1972).

Detection of the disease: The greening disease could be detected by preparing chromatograms from the bark and albedo of infected plants which show fluorescent spots due to presence of a fluorescent marker substance when viewed under ultraviolet lamp (Schwarz, 1965).

The greening agent was also detected successfully in the infected tissue by employing fluorescent antibody technique (Raychaudhuri *et al.*, 1972). Gamma globulins isolated from the antiserum prepared from the cultured greening mycoplasma-like organism, were labelled with fluorescein isothiocyanate which was used on hand sections of greening affected tissues for detection of the pathogen under fluorescent microscope.

Both these methods of detection are quite sensitive and can be used to detect the latent infections as well as they can be useful in distinguishing greening from foliar deficiency.

Control: Application of tetracycline antibiotics and B.P. an antibiotic produced by Hindustan Antibiotics has indicated encouraging therapeutic effect (Nariani *et al.*, 1971; Martinez *et al.*, 1970; Capoor and Thirumalachar, 1973). Infected budwood

subjected to moist hot air at 47°C for four hours or 43°C for six hours helped in inactivating the pathogen (Nariani *et al.*, 1973). Also subjecting potted budded greening affected seedlings (one to one and a half year old) to 38°–40°C for three weeks in a hot chamber freed the plants of greening symptoms.

Citrus Likubin

'Likubin' or 'yellow shoot' disease of citrus, prevalent in China and Taiwan, is similar to greening in India or stubborn in USA. Diseased leaves of affected trees are characterised by vein yellowing, swelling, corking and mesophyll chlorosis with green islands on the leaf surface. There is also premature defoliation, die-back of small twigs, formation of multiple abnormal flowers, decay of feeder roots and wilting of leaves of the diseased plants.

Transmission: The disease is transmitted by grafting. No vector is known.

Electron Microscopy: Numerous pleomorphic MLBs were detected in sieve elements of veins of diseased leaves (Su and Leu, 1972). More bodies were present in sieve tubes of old diseased leaves than the younger diseased leaves. Both ovoid forms 90 to 700 nm and filamentous forms measuring 40 to 90 200 to 2,000 nm were found. All the bodies were encased in a trilaminar unit membrane with a thickness of 14 to 24 nm.

Control: The diseased trees could be cured by heat or tetracycline therapy (Su and Leu, 1972). Achromycin solution at 100 ppm concentration completely checked the diseased development when budwood were dip-treated for fifteen hours. Likubin pathogen complex got inactivated by dipping the diseased scions in hot water, 48°C to 54°C for ten minutes.

Citrus Stubborn

This is prevalent in USA specially in naval oranges but affects all commercial varieties of citrus irrespective of root stock. This disease resembles the little leaf disease reported from Israel.

Symptoms: Disease is characterised by stunting of plants, excessive proliferation of axillary buds resulting in bunched upright growth of twigs, smalling and mottling of leaves. Diseased plants bear heavy normal and unseasonal flowers with excessive fruit drop. Fruits are deformed and acorn-shaped with insipid or bitter flavour and contain abnormal seeds.

Transmission: The disease is known to be spread by propagative material, bud wood or scion by grafting. No insect vector so far has been reported.

Electron microscopy: Several workers have shown the association of MLO with the infected plants (Lafleche and Bove, 1970b; Igwegbe and Calavan, 1970; Lafleche and Hauvat, 1972). Zelter *et al.* (1971) found MLB with little leaf affected citrus. Saglio *et al.* (1972) compared the ultra structure of greening and stubborn agents. Stubborn organisms generally possess 10 nm thick plasma membrane while that of greening is 20 nm.

Culturing: MLO isolated and cultivated from citrus stubborn has recently been named as *Spiroplasma citri*. This has already been discussed in the introductory portion of this chapter. Recently Daniels *et al.* (1973) reported the axenic culture of *Spiroplasma* from little leaf infected citrus plants.

Control: Control of stubborn mainly depends on cultural practices. The use of stubborn-free propagative materials from the disease free nurseries is most popular and effective.

Coconut Lethal Yellowing

This disease is prevalent in Jamaica, Haiti, Cuba, Togo, Ghana and Nigeria. Sometimes it comes in epidemic form.

Symptoms: Symptoms of coconut lethal yellowing are characterised by premature nut fall and blackening under the fruit calyx. Inflorescence gets brown in colour which start from the tip of the spikelets and progressing down to involve first male and then the female young flowers, thus finally the whole inflorescence becomes dark brown. Young heart leaves develop red brown rotting tips which gradually dry out. Older leaves or fronds turn yellow in colour. Infected palms usually die within three to six months after the appearance of the visible symptoms.

Transmission: The disease is suspected to be transmitted by a slow flying insect which has not been identified.

Electron microscopy: Bodies resembling mycoplasma were found in the sieve tube of affected trees. The bodies were highly pleomorphic bounded by 10 µm thick unit membrane. Both ovoid and filamentous forms have been observed (Plavsic-Banjac *et al.*, 1972; Beakhans *et al.*, 1972; Heinze *et al.*, 1972; Parthasarathy, 1974).

Control: McCoy (1972) found the remission of the disease by

tetracycline antibiotics. Antibiotics were applied by trunk injection method.

Cotton Small Leaf

The disease was first reported from India in 1944 and is prevalent in southern part of the country. It has also been described from Africa.

Symptoms: Disease is characterised by extreme reduction and stunting of the aerial parts of the plants. Leaves in the affected parts are lobed, deformed and often mottled. Sterility is very common phenomenon of the infected plants in which flowers remain very small and phylloid (Uppal *et al.*, 1944).

Transmission: The disease can be transmitted by grafting and not by sap inoculations. Vector of this disease is not yet reported.

Electron microscopy: A large number of pleomorphic MLB in the sieve tube elements of affected plants were reported by Capoor *et al.* (1972). Cousin *et al.* (1969) studied the virescence disease of cotton which is similar to small leaf disease from upper Volta in Africa. They found typical MLB with unit membrane, nuclear strands and ribosomes associated with this disease.

Eclipta prostrata Yellows Disease

It is prevalent in tropical and subtropical regions of the world and also occurs in India.

Symptoms: Chlorosis and phyllody are the chief disorder in affected plants (Padma *et al.*, 1973).

Transmission: The disease can be transmitted by grafting and not by sap inoculation.

Electron microscopy: Electron microscopy revealed pleomorphic to spherical mycoplasma-like bodies, varying from 200 nm to 600 nm in diameter in phloem parenchyma and sieve elements (Phatak *et al.*, 1974).

Hibiscus Rosa-sinensis Witches' Broom

It occurs in Brazil.

Symptoms: Affected plants show witches' broom, malformation and yellowing of the leaves.

Transmission: The disease is graft transmissible to healthy Hibiscus plants.

Electron microscopy: Pleomorphic bodies ranging in size from

150 nm to 425 nm, were found in phloem resembling mycoplasma (Marly Vicente *et al.*, 1974).

Opuntia Witches' Broom

Monstra variety of *Opuntia tuna* is widely propagated as an ornamental species.

Symptoms: Affected plant show stunted and anomalous growth of the stem.

Transmission: This disease has been reported to be graft transmissible (Uschdraweit, 1961). In nature the disease is suspected to be spread by a vector which has not yet been identified.

Electron microscopy: Casper *et al.* (1970), Maramorosch *et al.* (1972) reported the association of MLB with *O. tuna* var. *monstros* but not with *O. tuna*. MLB are present within the sieve cell of the phloem. When treated with tetracycline the bodies disappear from the cells.

Control: It has been shown that *O. tuna* var. *monstrosa* when treated with tetracycline shows the characteristics of *O. tuna*. So it has been concluded that *O. tuna* var. *monstrosa* is not a separate variety or species but was the name given to *O. tuna* when affected with witches' broom.

Papaya Bunchy Top

The disease is found in Florida, Puerto Rico, Santo Domingo, Haiti and Jamaica.

Symptoms: Affected plants are severely stunted, produce dwarfed and stunted internodes. Flowers produced on such plants fall off prematurely. A few fruits are produced which have an unpleasant taste.

Transmission: Disease is reported to be transmitted by the leaf hopper *Empoasca papaye* (Sein and Adsar, 1947) and by grafting and not by sap inoculations.

Electron microscopy: Association of MLB was reported by Storey and Halliwell (1969) with bunchy top disease.

Peanut Witches' Broom

Symptoms: This disease is prevalent in Indonesia, characterised by reduction of internodes, stimulation of axillary buds and formation of numerous small stiff leaves. In late stage of infection, gynophores begin to grow upward. Affected plants rarely produce fruits.

Transmission: Previously the disease was considered to be caused by a virus which was not sap transmissible. The disease is recently found to be transmitted by grafting as well as the leaf hopper, *Orosius argentatus* (Triharso, 1973).

Electron microscopy: Triharso (1973) also reported the association of MLB with the peanut witches' broom.

Potato Diseases

A number of mycoplasmal diseases have been described in potato. Some of these diseases produce a common symptom 'Hairy Sprout' in seed potato. Hairy sprout is mainly a storage disease and is common in the stores of Gangetic plains, Deccan plateau and Simla Hills of India. There are several reasons known for this disease, the sprouts emerging from affected tubers often show characteristic symptoms of yellows diseases of which marginal flavescence, potato purple top and potato witches' broom are very common. All the three diseases also occur in nature as such.

Potato Marginal Flavescence

This disease was first reported from Central Potato Research Institute, Simla, India (Nagaich and Giri, 1971) where the disease incidence was one to eight per cent. It has been estimated that under favourable condition loss in yield may be upto seventy per cent.

Symptoms: Symptoms of marginal flavescence consist of slight chlorosis on the margin of upper leaves. In some varieties chlorosis is accompanied by purplish pigmentation. The affected plants show stunting and produce cluster of small tubers attached to short and thin stolons.

Transmission: Disease can be transmitted by grafting as well as by the leaf hopper *Orosius albicinctus*, to the same host or to several other solanaceous hosts.

Control: The disease responds to tetracyclines, antiameobin, chloramphenicol and DPB, a coded antibiotic compound from Hindustan Antibiotic, treatments and gets temporarily cured.

Potato Purple Top

This was reported from India by Nagaich and Giri (1973a). Depending on variety the loss in yield varies from forty to seventy

per cent. Five to twenty-five per cent of the tubers from potato top roll (PTR) affected plants develop hairy sprout which reduces the market value of the seed tubers.

Symptoms: The diseased plants show purple pigmentation and rolling of basal parts of leaflets. Infected plants show severe stunting and growth of erect axillary branches. Root system is poorly developed and produces very short stolons. Mother tubers sometimes remain hard even at the time of maturity of the plant.

Transmission: The disease has been successfully transmitted through graft as well as leafhopper, *Orosius albicinctus* to the same host as well as to test plants like *Datura* spp. and *Lycopersicon esculentum*.

Electron microscopy: Nagaich and Giri (1973b) showed the presence of MLB in phloem cells of the diseased plants.

Control: Antiamoebin, chloramphenicol and tetracycline compounds have curative effect on the disease.

Potato Witches' Broom

Mycoplasmal etiology of the disease was first suspected by Doi *et al.* (1967).

Symptoms: Affected plants develop numerous slender and weak stem, bearing small leaves. Such plants are extremely stunted with bushy appearance and produce a large number of very small tubers.

Transmission: Disease can be transmitted by grafting as well as the leafhopper *Orosius albicinctus*.

Electron microscopy: The sieve tube and phloem parenchymatous cells of witches' broom affected stem and leaves of potato plants, showed the association of MLB (Doi *et al.*, 1967). The bodies were pleomorphic (80–800 nm) and similar to mulberry dwarf organism. Association of MLB has been confirmed later on by Broak *et al.* (1969) and Harrison and Roberts (1969). These authors found largest bodies upto 1000 nm and showed the presence of filamentous forms.

Control: The tetracycline research group of Japan (1968, 1969) and Brvak *et al.* (1969) found suppression of disease symptoms by tetracyclines but no permanent cure was achieved. Due to phytotoxicity effect the antibiotics were considered to be of limited use.

Rice Yellow Dwarf

The disease was first recorded in Japan in 1910. Now it is known

to occur in many East and Southeast Asian countries like Taiwan, Philippines, Thailand, Malaysia, Bangladesh and Sri Lanka, and in some parts causes considerable damage. In India the disease has been recorded in Andhra Pradesh, Bihar, Delhi, Orissa and West Bengal (Raychaudhuri *et al.*, 1967b) and Karnataka (Govindu *et al.*, 1968).

Symptoms: Yellow dwarf is characterised by stunting and profuse tillering of the plants. These tillers bear uniformly chlorotic pale green to pale yellow leaves. Chlorotic symptoms first appear on the youngest emerging leaves.

Early infected plants die prematurely while the late infected plants may survive but remain sterile. Sometimes late infected plants do not show any symptoms on the standing crop but produce visible symptoms on ratoon crops on cut stubbles.

Transmission: Three species of leaf-hoppers, *Nephotettix cincticeps*, *N. nigropictus* and *N. virescens* have been reported as vectors of this disease. Shinkai (1962) studied the transmission of the disease in detail. The hoppers acquire the causal agent by feeding of affected plants for one to three hours. There is a long incubation period of twenty-five to thirty days in the insect and twenty to thirty-nine days in plant. Once infective, the vector can transmit the agent to new plants throughout their life and it is not found to be transovarially transmitted.

Alopecurus aequalis and *Glyceria acutiflora* were found to be the alternate host of this disease (Shinkai, 1962). Hopper were found to over winter on *A. aequalis* or ratoon plants.

Electron microscopy: Nasu *et al.* (1967) found association of MLO in the sieve elements of infected plants as well as epithelial cells and salivary glands of infective *N. cincticeps*. Shikata *et al.* (1968, 1969a) reported similar findings in Philippines while Sugiura *et al.* (1968) and Plavsic-Banjac (1973a) reported the association of MLB in the yellow dwarf material collected in India.

The pleomorphic bodies are surrounded by unit membrane and contained ribosomes and DNA like strands. Most of the bodies were found in mature sieve elements. Companion cells were devoid of MLO.

Control: Attempts have been made in J. van to control the insect vectors of the disease by aerial application of insecticides over large areas. Seed dressing with carbofuran and propoxure can protect rice seedlings from the leafhoppers, the common vectors

for both tungro virus and rice yellow dwarf (Mitra *et al.*, 1970).

Safflower Phyllody

This disease was reported from Israel (Klein, 1970).

Symptoms: Affected plants are characterised by bushy growth of the plant. Primary shoots produce many very thin branched secondary shoots. These secondary shoots bear very small yellowish leaves. Phyllody of flowers is a common phenomenon. Sometimes flowers produced on the infected plants have abortive seeds.

Transmission: In nature the disease was found to be transmitted by *Neolaliturus fenestratus*. In laboratory this vector could transmit the disease agent to several members of family compositae as well as *Vinva rosea*.

Electron microscopy: Characteristic MLB were detected (Klein, 1970) in ultrathin sections of phloem cells of the *Vinca* plants infected with safflower phyllody. Under high magnification, bodies were surrounded by typical unit membrane, inside which DNA-like and ribosome-like structures were visible.

Salix (Willow) Witches' Broom

Witches' broom disease of *Salix rigida* found sporadically in southern New Hampshire, New York and Massachusetts. In India disease is seen in Simla Hills and Kashmir valley.

Symptoms: Disease is characterised by the development of numerous axillary shoots. These axillary shoots are usually erect, thin and spindle shaped bearing small leaves. Graft induced plants were characterised by initial vein clearing followed by breaking of dormancy of axillary buds.

Transmission: The disease is graft transmissible. No insect-vector has been identified.

Electron microscopy: Pleomorphic MLB were detected in sieve tube elements of diseased leaves (Holmes *et al.*, 1972). The bodies were delimited by triple-layered unit membrane and contained DNA and ribosome particles.

Sandal Spike

Sandal spike was first reported by McCarthy from Coorg District of Karnataka State in India, in 1899 (Barber, 1903). Now the

disease is progressively spreading throughout the whole southern part of Karnataka as well as border districts of neighbouring states. The disease takes a heavy toll and is threatening the whole sandal industry (Varadaraja Iyengar, 1969). At Government Sandal Wood Factory, Mysore, it has been estimated that fifteen, forty year old healthy sandal trees produce approximately one ton of sandal wood, while the equivalent amount of wood is produced by more than 300 spiked trees.

Symptoms: Two type of symptoms are recognised, the most common "Rosette spike" is characterised by severe reduction of leaf size and shortening of internodes. As the disease advances the new leaves emerging become smaller and smaller. Leaves on the affected twigs have a tendency to stand out stiffly like spikes. In very advanced stage leaves become yellowish and finally reddish brown before the death of the plant. Symptoms may appear in the whole tree or in isolated branches. The trees usually die within two to three years after the appearance of the first visible symptom. Phyllody of flower is rare and the affected twigs remain in vegetative phase only.

Other type of symptom, "Pendulous spike" is quite rare and was first described by Venkata Rao and Iyengar (1934). The characteristic feature of Pendulous spike is that the individual infected shoots show continuous apical growth and assume a drooping habit. Axillary buds usually remain dormant.

Transmission: The disease is graft transmissible. Dijkstra and Lee (1972) reported that the disease could be transmitted through dodder. *Mopnia alhimaculata* and *Jasus indicus* have been reported to be the vectors of sandal spike (Ramaswamy and Griffith, 1941). however, the transmission of the disease with these vectors requires confirmation.

Electron microscopy: Presence of MLB was first shown in the phloem tissue of spiked plant by Varma *et al.* (1969) which was almost simultaneously confirmed by Dijkstra and Ic (1969) and Hull *et al.* (1969). Plavsic-Banjac *et al.* (1973b) found MLO in the phloem elements of naturally infected or as well as in the graft-induced spiked plant of sandal.

Control: After determining the nature of the causal agent, attempts were made to control the disease by chemotherapy. Tetracycline compounds as in other yellows diseases and the systemic fungicide benlate were effective against the disease (Raychaudhuri *et al.*, 1972.

1973). Combination treatments of benlate with tetracyclines when applied by 'girdling' method, were much more effective.

Sugarcane Grassy Shoot

This is one of the most serious diseases of sugarcane widespread specially in Maharashtra State of India from where this was first reported (Vasudeva, 1955). The disease is also prevalent in Andhra Pradesh, Bihar, Madhya Pradesh, Tamilnadu and Uttar Pradesh. The disease is also reported from Thailand.

Symptoms: The disease causes production of numerous tillers from the base of the mother plants. Bunches of these tillers bear pale yellow, thin narrow leaves resembling grasses. These stunted tillers are with reduced internodes. In severe cases there is no cane formation.

Transmission: Grassy shoot disease is reported to be sap and aphid transmitted (Chona *et al.*, 1960). Three known vectors are *Aphis ideosacchari*, *A. sacchari* (*Longiunguis sacchari*) and *A. maidis* (*Rhopalosiphum maidis*). The disease could be transmitted to *Sorghum vulgare* which serves as collateral host.

Electron microscopy: Corbett *et al.* (1972) and Rishi *et al.* (1973) published the electron micrographs of the ultrathin sections of grassy shoot affected plants thereby showing the presence of typical MLB in the phloem cells. Stages of budding and binary fission were also observed (Rishi *et al.*, 1973).

Control: Singh *et al.* (1973) found temporary remission of the disease symptom with tetracycline treatment. Cent per cent control of grassy shoot disease both in plant crop and in ratoon crop can be obtained by exposing the seed canes to hot air, 54°C for eight hours in a specially designed air tight chamber for the treatment of canes (Singh, 1968; Singh *et al.*, 1973).

Sugarcane White Leaf

The disease was first observed in Taiwan in 1958 (Ling and Chuang-Yang, 1962). The disease is quite different from grassy shoot or albino diseases of sugarcane reported from India.

Symptoms: Though the disease is quite different from grassy shoot disease in mode of transmission, etc., symptoms are more or less similar. Diseased plants may easily be identified by the disappearance of green colour from the leaves which are narrow, chlorotic and whitish yellow in colour. The affected

plants are markedly stunted with thin internodes.

Transmission: The disease is transmitted by the leaf hopper vector *Epitettix hiroglyphicus* and not by aphids which transmit grassy shoot (Matsumoto *et al.*, 1969).

Electron microscopy: MLO were detected in the leaves of sugar-cane plants affected with white-leaf disease (Shikata *et al.*, 1969b). MLB sometimes appeared accumulated and seemed to plug the phloem cells.

Culturing of organisms: Lin *et al.* (1970) reported the isolation and cultivation of mycoplasmas from this disease. They also claimed to reproduce the disease symptoms in healthy plants by mechanical inoculation.

Control: The disease can be checked by immersion of the diseased seed sets in terramycin, achromycin or aureomycin (Shikata *et al.*, 1969b).

Sesamum Phyllody

The disease is quite common in sesamum growing areas. Though the disease is known in India for a very long time, Pal and Pushkar-nath (1935) reported it as graft-transmitted virus disease.

Symptoms: Vein clearing, stimulation of axillary buds resulting in profuse branching, reduction of leaf size, shortening of internodes and phyllody of flowers are some of the characteristic symptoms of this disease. All the floral plants are transformed into green leaf like structures resulting into complete sterility.

Transmission: The disease is transmitted by the vector *Orosius albicinctus* (Vasudeva and Sahambi, 1955). Biology, bionomics and the pathogen-vector relationship has been studied in details by Sahambi (1970) and, Bindra and Singh (1970). Depending on the environmental conditions specially the temperature the incubation period of the pathogen in the leaf hopper varies from fifteen to sixty-three days and thirteen to sixty-one days in the host. The disease could be transmitted through vector or by grafting to a number of plants in field crops, ornamentals and weeds. Vectors can overwinter in all stages and present in nature in small or greater in number around the year.

Electron microscopy: Electron microscopic examinations of the diseased material collected in the upper volta in Africa revealed the presence of pleomorphic MLO by Cousin *et al.* (1970). Choopanya (1973) also reported the association of mycoplasmal

with sesamum phyllody in Thailand.

Control: Tandon and Banerjee (1968, Bindra and Singh (1970) attempted to check sesamum phyllody by controlling its vector with insecticides. They did not, however, mention the extent of control of the disease.

Rickettsia-Like Organisms With Plant Diseases

Since 1967 when the plant pathologists were exploring the possible involvement of mycoplasmas as pathogens for yellows diseases of plants by studying ultrathin sections and chemotherapy of the affected plants, a few workers suspected Rickettsia as causal agent for some of the diseases belonging to the yellows group, namely, stunted dodder (Giannotti *et al.*, 1970), clover clubleaf (Windsor and Black, 1973b), Pierce's disease of grapevines, alfalfa dwarf (Goheen *et al.*, 1973) and Phony peach (Hopkins *et al.*, 1973; Nyland *et al.*, 1973) diseases. Under electron microscope typical rickettsia-like bodies are found in the lumen of and between the xylem vessels. The organisms are pleomorphic rod-shaped bacterium $0.4 \times 3-2\mu$, delimited by a triple cell wall. Cytoplasm contained electron dense organells and surrounded by cytoplasmic membrane.

Some of the diseases get suppressed with penicillin and tetracycline antibiotics (Windsor and Black, 1973a) or heat treatment (Goheen *et al.*, 1973).

TABLE 1

DIFFERENCE BETWEEN MYCOPLASMA AND ITS RELATED ORGANISMS
(MODIFIED FROM MOULDER, 1968)

<i>Organism</i>	<i>Nature of nucleic acids</i>	<i>Mode of reproduction</i>	<i>Growth in cell-free medium</i>	<i>Generation of metabolic energy</i>	<i>Rigid cell wall</i>
Viruses	DNA or RNA	Synthesis of sub-units, then assembly of complete virus	No	No	No
Rickettsiae	DNA and RNA both	Fission of parental cell	No	Yes	Yes
Chlamydiae	DNA and RNA both	Fission of parental cell	No	No	No (large cells) Yes (small cells)
Bacteria	DNA and RNA both	Fission of parental cell	Yes	Yes	Yes
L-forms of bacteria	DNA and RNA	Fission of parental cell	Yes	Yes	No
Mycoplasmas	DNA and RNA both	Fission of parental cell	Yes	Yes	No

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Index

- Abaca**
 bunchy top 114
 mosaic 114
- Abrasives** 13
- Adelges abietis*** 125
- Abutilon** mosaic 263
- Acalypha*** yellow mosaic 17, 263
- Aceria ficus*** 18
- Achyranthes*** mosaic 264
- Acroclinium*** mosaic 176
- Adjuvant** 43
- Agathia albidula*** 226
- Agalliana ensigera*** 225
- Ageratum*** yellow vein mosaic 264
- Agglutination** 46, 48, 49
- Aleurotrachelus socialis*** 248
- Alfalfa** mosaic on broad bean 28, 53
- Almond** yellow vein 126
- Amorphophallus*** mosaic 224
- Anaphylaxis** 45
- Antibody** 43-7, 56
- Antigen** 43-7
- Antiserum** 46-9, 55
- Antiviral chemicals** 62
- Aphis***
 craccivora 87, 122, 162, 163, 164,
 169, 178, 200, 201, 211, 212,
 213, 216, 218, 228, 232, 235,
 236, 241, 243, 246, 253, 265
 evonymii 153, 200
 fabae 158
 ferruginea-striata 179
 gossypii 95, 115, 121, 129, 130,
 137, 138, 141, 143, 154, 156,
 178, 180, 181, 186, 196, 200
 medicoginis 143
 maidis 95
 nerii 173
 pseudobrassicae 178
 rumicis 130, 143, 174, 179, 183
 malvae 143
- Apple**
 bunchy top 126
 leaf pucker 126
 little leaf 127
 mosaic 127
 rubbery wood 270
 star-crack 8, 128
- Apricot**
 necrosis leaf roll 128
 yellow mosaic 128
- Arabis*** mosaic 19, 82
- Areca nut*** yellow leaf 189
- Artichoke** mosaic 128
- Aster yellows** 17, 85
- Bajra (*Pennisetum typhoides*)**
 mosaic 159
 streak 159
- Banana**
 bunchy top 83, 129
 mosaic 16, 83, 129
- Barley**
 chlorotic spot 55
 false stripe 53, 54
 mosaic 15, 16, 49, 92

- stripe mosaic 49, 53-5, 75, 93
- yellow dwarf 16, 27, 93
- yellow dwarf mosaic 92, 93
- Bean (*Phaseolus vulgaris*)**
 - atypical mosaic 226
 - mosaic 15, 16, 38, 53, 83, 226
 - yellow mosaic 10, 227
- Beet**
 - curly top 225
 - purple leaf 225
 - yellow vein 226
- Bemisia** sp. 141
 - goldingi* 116
 - gossypiperda* 116, 186
 - inconspicus* 116
 - tabaci* 17, 116, 117, 130, 142, 178, 179, 184, 200, 202, 204
 - tuberculata* 248
- Bermuda grass mosaic 264
- Berseem mosaic 121
- Bhindi (*Abelmoschus esculentus*)
 - yellow vein mosaic 6, 83, 230
- Boerhavia** mosaic 265
- Bottle gourd mosaic 49, 69, 73, 74, 76, 237
 - (Same as cucumber green mottle)
- Bougainvillea mosaic 173
- Brachycaudis helichrysi* 106
- Brevicoryne brassicae* 176, 179
- Brinjal**
 - little leaf 17, 270
 - mosaic 84, 231
- Broad bean mosaic 9, 10, 41, 73, 228
- Brome grass mosaic 27
- Budwood certification 83
- Butter cup mosaic 173
- Cabbage blackring spot** 9, 232
- Cacao**
 - mosaic 193
 - mottle leaf 190
 - swollen shoot 8, 17, 18, 83, 189
 - swollen shoot strain A 190, 192 (or red mottle)
 - swollen shoot strain B 191, 192 (or vein clearing)
 - swollen shoot strain D 191
 - yellow mosaic 193
- Calotropis* mosaic 153
- Canna**
 - mosaic 174
 - mottle 174
- Cape gooseberry
 - mosaic 130
 - leaf curl 130
- Cardamom**
 - large (*Amomum subulatum*)
 - chirke (mosaic streak) 16, 48 221
 - foorky 16, 67, 221
 - small (*Elettaria cardamom*)
 - katte 16, 220
 - marble 220
- Carneiocephala fulgida* 139
- flaviceps* 139
- Cassava mosaic 49, 193
 - stem lesion 194
- Cauliflower mosaic 7, 233
- Centrifugation**
 - density gradient 36
 - differential 40
 - rate zonal 37
- Chick pea mosaic 210
- Chilli**
 - leaf curl 233
 - mosaic 16, 18, 61, 62, 71, 72, 74, 84, 234
 - mosaic (necrosis) 235
 - pepper vein banding mosaic 236
 - PVX 236
 - yellow vein mosaic 83
- Chlorotic lesion 6
- Chrysanthemum aspermy 85, 175
- Cicadulina*
 - bipunctella 105
 - mbila 16, 96, 159
 - nicholsi 96
 - zeae 96
- Cinaropsis pilicornis* 125
- Circulifer tenellus* 16
- Citrus**
 - blind pocket 135
 - concave gum 135
 - crinkly leaf 131
 - exocortis 132
 - greening 85, 271
 - infectious variegation 133

- leaf curl 131
- leaf mottle 132
- likubin 273
- psorosis 134
- psorosis A 134
- seedling yellows 137
- stubborn 8, 273
- tristeza 85, 135
- vein enation 137
- woody gall 7, 137
- xyloporosis 138
- Clover
 - vein mosaic 9
 - wound tumor 29
- Coconut
 - cadang cadang 195
 - lethal yellowing 274
 - root wilt 195
- Coffee
 - blister spot 196
 - ring spot 196
- Coffee senna ring spot mosaic 153
- Common bean
 - enation mosaic 229
 - yellow mosaic 229
- Complement fixation 44
- Corn stunt 17
- Cotton
 - anthocyanosis 115
 - leaf crumple 115
 - leaf curl 116
 - leaf mottle 116
 - little leaf 275
 - veinal mosaic 117
- Cowpea
 - mosaic 27, 53, 54, 56, 57, 62, 63, 64, 73, 75, 76, 210, 211
 - necrosis 214
 - chlorotic spot 49
 - vein banding 214
 - yellow mosaic 214
- Cucumber mosaic 14, 20, 36, 39, 72, 73, 76, 87
- Cucumis mosaic virus I 236
 - mottle virus 8
 - yellow vein mosaic 241
- Cuerna occidentalis* 139
 - yuccae* 139
- Cuscuta*
 - subinclusa* 20, 135, 136
 - californica* 20
 - campestris* 20, 205
 - compacta* 135
 - reflexa* 100, 136, 173
- Dactynotus* sp. 94
- Dahlia mosaic 49, 175
- Dalbulus maidis* 17
- Datura*
 - distortion mosaic 39, 154
 - enation mosaic 154
- Diaphorina citri* 132
- Dolichos enation mosaic 27, 48, 49, 229
- Double bean yellow mosaic 229
- Draculacephala*
 - californica* 139
 - minerva* 139
 - naveboraccus* 139
 - orassicernis* 139
- Eclipta* yellows 275
- Empoasca devastans* 274
 - papayae* 276
- Endria inimica* 80
- Eriophyid mite 18
- Euphorbia* mosaic 265
- Fig mosaic 18, 138
- Frankliniella*
 - insularis* 17, 255
 - moultoni* 255
 - occidentalis* 255
- Ferrisia virgata* 199
- Garlic mosaic 244
- Grafting 12
 - approach or inarch 13
 - bud 12
 - cleft or wedge 12
 - core 13
 - patch or bark 13
- Grapevine
 - asterioid mosaic 140
 - fan leaf 19, 140

- mosaic 140
- pierce's disease 139
- yellow mosaic 19
- Graphocephala cythra* 139
- Groundnut (peanut)
 - bud light 166
 - bud necrosis 167
 - bunchy top 165
 - chlorosis 16, 165
 - chlorotic spot 167
 - clump 164
 - cushion 163
 - helper virus 164
 - mosaic 162
 - mottle 163
 - ring mottle 165-6
 - ring spot 163
 - rosette 163
 - shoot necrosis 167
 - spotted wilt (or chlorosis) 166
 - witches' broom 276
- Guar (*Cyamopsis tetragonoloba*)
 - necrosis 243
- Heliochara delta* 139
- Henbane mosaic 49, 266
- Hibiscus mosaic 176
- Himonas phycitis* 17
- Hippeastrum* mosaic 177
- Homolodisca liturata* 139
- Hordina circellata* 139
- Hyperomyzus lactucae* 245
- Hyperplasia 8
- Hypertrophy 10
- Hypoplasia 8
- Inclusions 9, 10, 56
 - amorphous 9
 - cellular 57
 - crystalline 9, 10
 - cytoplasmic 56
 - intranuclear 56
 - tubular 56
- Jasmine chlorotic ring spot 177
- "Jatropha"
 - leaf distortion 178
 - mosaic 177
- Javesella pellucida* 96
- Jute
 - chlorosis 117
 - yellow mosaic 117
- Kymograph 45
- Laodelphox striatellus* 96
- Lettuce
 - big vein 19
 - mosaic 53, 244
 - necrotic yellows 245
 - yellow mosaic 245
- Lipaphis erysimi* 173, 200
- Local lesion 6, 28
- Longidorus elongata* 19
 - attenuatus* 19
- Longiunguis sachari* 158
- Lucerne mosaic 121
- Macrosiphoniella sonbornii* 228
- Macrosiphum*
 - avenae* 94
 - diurhodum* 94
 - gei* 176
 - granarium* 94, 95, 157
 - plsi* 121, 130, 179
 - solanifolii* 143, 174
 - sonchi* 143
- Macrosteles fascifrons* 131
- Madagascar mosaic 153
- Maize
 - dwarf mosaic 27, 94
 - mosaic 84, 95
 - rough dwarf 96
 - streak 96
- Malvavistis* leaf curl 179
- Malvastrum* yellow vein mosaic 266
- Marigold mosaic 178
- Megoura viciae* 227
- Melanoplus differentialis* 18
- Micromyzus*
 - formosanus* 143
 - kalimpongensis* 222
- Microscopy
 - electron 55
- Mopnia albimaculata* 281
- Mulberry

- mosaic 140
- yellow net vein 141
- Mung yellow mosaic 57
- Mustard (chinese sarson)
 - mosaic 168
- Myzus circumflexus* 174, 179
 - convolvuli* 176
 - persicae* 130, 138, 141, 143, 144, 154, 156, 157, 173, 174, 175, 179, 180, 183, 185, 200
- Nasturtium
 - mosaic 179
 - ring spot 179
- Necrosis 8
- necrotic lesions 6
- Neolaliturus fenestratus* 280
- Neokolla grothica* 139
- Nephotettix* 103
 - apicalis* (same as *nigropictus*) 16, 103
 - bipunctatus* 106
 - cincticeps* 106
 - malayanes* 103
 - nervus* 103
 - virescens* (same as *impicticeps*) 16, 103
- Nilaparvata lugens* 99
- Oat red leaf 97
- Olpidium brassicae* 19
- Onion yellow dwarf 243
- Opuntia* witches' broom 276
- Orchid mosaic 180
- Orosius* sp. 165
 - albicinctus* 276
 - argentatus* 165, 277
- Panax ring spot 180
- Papaya
 - bunchy top 276
 - leaf curl 142
 - mild mosaic 144
 - mosaic 143
 - ring spot 141
- Passiflora* yellow vein mosaic 144
- Pea
 - leaf roll 210
 - mosaic 16, 245
- Peach
 - little peach 26
 - mosaic 145
 - yellows 26
 - rosette 20
- Pear mosaic 145
- Pentalonia nigronervosa* 114, 129, 158
- Perigrinus maidis* 160
- Perkinsiella vastatrix* 197
- Periwinkle
 - leaf curl 74, 181
 - mosaic 180
- Petunia mosaic 49, 181
- Phascolus atropurpureus* mosaic 122
- Phascolus aureus* yellow mosaic 17, 215
- Phaseolus longipedunculatus* 246
- Phlox mosaic 182
- Pigeon pea
 - mosaic 215
 - sterility 18, 215
- Pigweed (or *Amaranthus*) mosaic 224
- Pleuropneumonia like organisms 268
- Plum
 - creamy white spots 145
 - enation mottle 145
 - leaf roll 145
 - line pattern 145
 - ring spot and shot hole 147
 - pox 8
- Polyomyxa graminis* 19
- Poplar mosaic 124
- Potato
 - aucuba mosaic 247
 - calico 247
 - foliar necrosis 247
 - leaf roll 7, 9, 10, 26, 27, 86, 87, 248
 - leaf curl 248
 - marginal flavescence 288
 - mild mosaic 249
 - mop top 20
 - purple top 8, 277
 - marginal flavescence 277
 - rugose mosaic 249

- severe mosaic 250
 supermild mosaic 250
 top necrosis 9
 virus A 48
 virus S 48
 virus X 8, 18, 19, 36, 61, 62, 63,
 69, 70-5, 82
 virus Y 9, 15, 29, 48, 49, 62,
 63, 70-4, 87
 witches' broom 278
 yellow vein 251
 Precipitation 44
 Primula
 mosaic 183
 mottle 183
Pseudococcus citri 190, 191
 njalensis 191
 Pumpkin mosaic 238

 Radish mosaic 69, 74, 87, 251
 Ragi (*Eleusine coracana*)
 mosaic virus I 157
 mosaic virus II 157
 Eleusine mosaic virus 158
 Rai (*Brassica juncea*)
 mosaic 168
 Raspberry mosaic 147
 ring spot 147
Rauvolfia Serpentina
 bunchy top 155
 Reccilia (see *inazuma*) *dorsalis*
Rhopalosiphum maidis 92, 94, 115,
 141, 157, 158, 159
Rhopalosiphum nymphicae 115
 padi 94
 prunifoliae 115, 179
 pseudobrassicae 122
 rufiabdominalis 157
 Rice
 dwarf 16, 98
 grassy stunt 99
 hoja-blanca 99
 mosaic 100
 orange leaf 100
 transitory yellow 101
 tungro 16, 84, 101
 mentek penyakit merah 106
 yellow dwarf 278

 Rose
 yellow mosaic 183
 yellow vein mosaic 184
 Runner bean mosaic 230

Sacciphantes abietis 125
 Safflower mosaic 169
 phyllody 280
 Salix witches' broom 280
 Sandal
 leaf curl mosaic 124
 spike 280
Schizaphis cyperi 158
 granarium 94, 198
 Sesamum
 leaf curl 170
 mosaic 170
 phyllody 8, 283
 Shape of virus 1, 2
 Shoefflower
 leaf curl 184
 line pattern 184
Sida carpinifolia
 infectious chlorosis 155
 Spinach blight 26
 Snake gourd mosaic 49, 237
 Soapwort leaf curl 185
Sogata
 cubens 99
 oryzicola 99
Sogatella sp. 158
 Solanum phasianum mosaic 155
 Sorghum chlorosis 160
 Southern bean mosaic 53
 Soybean mosaic 55, 74, 216
 yellow mosaic 217
Spiroplasma citri 274
Spongospora subterranea 20
 Spruce virosis 125
 Squash mosaic 18, 55
Stephanitis typicus 195
 Sugar curly top 8, 20, 28
 Sugarcane
 fiji 197
 grassy shoot 282
 mosaic 197
 ratoon stunting 198
 streak 199

- white leaf 282
- Sunflower mosaic 185
- Sunn hemp mosaic 17, 39
- Southern sunn hemp mosaic 12, 39, 49, 61, 70, 72
- Sweet potato mosaic
 - virus A 252
 - virus B 252
- Synchytrium endobioticum 19
- Tea
 - yellow mosaic 199
 - phloem necrosis 84, 199
- Therapy
 - chemo 86
 - heat 85
- Tetraneura nigrabdominalis* 158
- Thevetia* leaf curl 185
- Trialeurodes abutilonia* 116
- Trichodorus Pachydermus* 19
- Thrips tabaci* 255
- Tobacco
 - broken ring spot 200
 - distortion 200
 - etch virus 9, 10, 38, 201
 - leaf curl 7, 17, 29, 84, 86, 202
 - mosaic or TMV 7, 9, 15, 18, 20, 26, 28-30, 36, 38, 39, 48, 53-7, 61-4, 66, 70, 72-5, 86, 87, 202
 - necrosis 7, 19, 36, 49
 - ring spot 7, 36, 53, 204
 - streak 204
 - rattle 19
 - stunt 19
 - yellow net 204
- Tomato
 - aucuba mosaic 252
 - black ring 19, 82
 - black ring spot 253
 - bunchy top 253
 - bushy stunt 10, 20, 38, 75, 86, 87
 - condensed top disease 254
 - curly top 256
 - enation leaf curl 255
 - leaf curl 83, 86 254
 - spotted wilt 17, 26, 28, 255
 - streak 86
- Tomasis liburata* 199
- Tori (*Luffa acutangala*) mosaic 49, 247
- Toxoptera *aurantii* 199
- citricidus* 136, 137, 138
- granarium* 158
- Turnip crinkle 18, 19, 256
- yellow mosaic 18, 19, 29, 38
- rosette 18
- Urid (*Phaseolus mungo*)
 - leaf crinkle 217
 - mosaic 217
 - yellow mosaic 217
- Vegetable marrow
 - mosaic 239
 - yellow mosaic 240
- Viroid 2
- Virus inhibitors 69
- Virus
 - persistent 15
 - non-persistent 16
- Watermelon
 - mosaic 39, 49, 242
 - vein banding mosaic 242
- Wheat
 - mosaic 107
 - streak mosaic 106
 - (see also 'chirke' disease of large cardamom)
- Whitefly 17
- X-bodies 9
- Xiphinema* sp. 19
- americanum* 204
- diversicaudatum* 19
- index 19, 140
- Zinnia
 - leaf curl 17, 186
 - mild mottle 186
 - mosaic 69, 74, 185